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# TEXAS State Journal of Medicine

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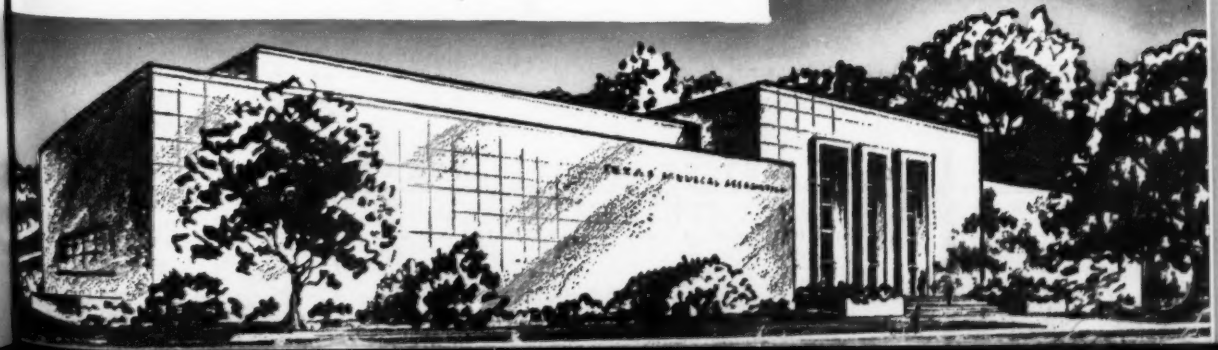

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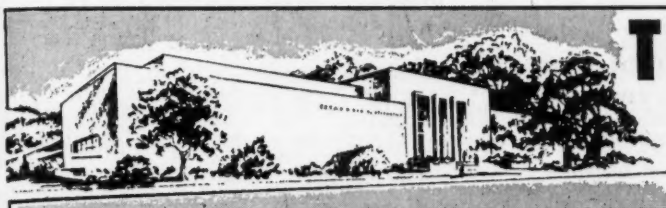
Ninetieth President, Texas Medical Association

SAN ANTONIO, TEXAS

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## J. LAYTON COCHRAN

The ninetieth president of the Texas Medical Association, Dr. J. Layton Cochran, San Antonio, accepted the gavel of office from his predecessor, Dr. F. J. L. Blasingame, during the closing ceremony of the 1955 annual session in Fort Worth on April 27.

Dr. Cochran was born June 12, 1900, in Ozona, the son of Daisy May (Harrell) and William A. Cochran.

In Sanderson, Dr. Cochran received his preliminary education, after which he was graduated from the University of Texas in 1921 and earned his doctor of medicine degree from the University of Texas Medical Branch, Galveston, in 1924. The following year, he interned at the Robert B. Green Memorial Hospital in San Antonio, and then he began general practice in that city.

A leader in the organized activities of the medical profession, Dr. Cochran is a past president of the Bexar County Medical Society and served as district councilor for five years. He is a member of the Texas, Southern, and American Medical Associations; the Southwestern Surgical Society; and the Texas Academy of General Practice, of which he was president-elect of his chapter in 1954. He resigned this post before succeeding to the presidency so he could devote more time to the Texas Medical Association this year. He was secretary of the Association's Section on General Practice in 1950 and its chairman in 1951. He was chairman of general arrangements for the annual session in 1949. Dr. Cochran also is a past president of the International Medical Assembly of Southwest Texas. Serving the more unfortunate members of his community, Dr. Cochran has headed his county hospital staff and has been chief of gynecology at the hospital, the Robert B. Green. He also has been a leader of the American Cancer Society's District 5.

Not only have his professional colleagues looked to him for leadership but also he has served as chairman of the board of the Travis Park Methodist Church and as its lay leader.

Dr. Cochran finds time during his busy schedule to take part in many social organizations in his home city. As a patron of the gala Battle of Flowers, he is a member of the Fiesta Association and the Texas Cavaliers, and was chosen King of the Fiesta in 1939. He also is a member of the Conopus Club, the Oak Hills Country Club, and the San Antonio German Club. He counts golf as his hobby.

Serving in the Army Medical Corps during World War II, Dr. Cochran was a surgical staff member of the Fort Sam Houston and Camp Wolters Station Hospitals, then was transferred to the station hospital in Fort Amador, Panama, where he was chief of the surgical staff for twenty months. After a short term of duty in the United States, he was assigned to the Ninety-Seventh Evacuation Hospital in the European Theater. During his service there, he was decorated for gallantry before the enemy at the Battle of the Bulge. He served actively from 1940 until 1945, and is now a lieutenant colonel in the honorary reserve.

Dr. Cochran is not married. He lives with his mother and his sister, Miss Ruby Mae Cochran, San Antonio. He also has a brother, Carl B. Cochran, Sanderson.

The new President has given "unselfish and devoted service—to his country, to his church, to his community, and to his profession," as was pointed out in the speech last year before the House of Delegates nominating him as President-Elect of the Texas Medical Association. Modest and affable, Dr. Cochran speaks his convictions frankly and stands firm when convinced he is right. He is a man whom his fellows can respect, and respecting, can join wholeheartedly in advancing the causes of the Texas Medical Association this year.

## BIGGEST AND PERHAPS BEST

The largest annual session in Texas Medical Association history was held in Fort Worth the week of April 24. The "largest," while applying specifically to the total registration of 2,685, also is applicable to the array of scientific, business, and social activities held during the session.

It was agreed generally that the scientific program sponsored by the Association and related specialty groups, featuring thirty-one special speakers in addition to local essayists, twenty-five well attended refresher courses, scheduled motion pictures, and strong general, section, and specialty sessions, was of a caliber to satisfy the most exacting and a pattern worth following next year.

The House of Delegates worked diligently, adopting amendments to the Constitution and By-Laws to eliminate the word "white" in the list of qualifications for membership in the Association. A plan to strengthen the organization by requiring a two year probationary membership with an indoctrination program and another to make it compulsory for active members to attend a minimum of 30 per cent of county medical society meetings to remain in good standing, were heard but will not be voted on until next year. Consideration was given to a variety of subjects in the public eye —Salk vaccine, fluoridation of public water supplies, civil defense, extension of social security, care at Veterans Administration hospitals, corporate practice of medicine—as well as other projects being carried on by the Association's committees. Details on action taken relative to these and other subjects will appear in the June JOURNAL.

Among the pleasant duties of the House was the election to places of honor of a number of physicians. Dr. Milford O. Rouse, Dallas, was named President-Elect to succeed Dr. J. Layton Cochran of San Antonio, who accepted the gavel of office from the outgoing President,

Dr. F. J. L. Blasingame of Wharton. Dr. S. W. Thorn, Houston, was chosen Vice-President; Dr. Hobart O. Deaton, Fort Worth, and Dr. Charles P. Hardwicke, Austin, Speaker and Vice-Speaker, respectively, of the House. Selected as General Practitioner of the Year was Dr. R. W. Holton of Terrell. Dr. O. M. Marchman, Dallas, was elected to emeritus membership in the Association, and Dr. Charles S. Venable, San Antonio, was nominated for emeritus membership, a final vote being delayed until next year in accordance with the Constitution.

An overflow crowd of about 1,300 attended the dinner dance honoring President Blasingame, and a sizable number of doctors and their wives stayed through the final luncheon to hear Leonard E. Read. The alumni, fraternity, and specialty parties were reported to be well attended, and such entertainment as the guided tours planned by the host society and auxiliary drew an unexpected turnout.

The Woman's Auxiliary, meeting simultaneously with the Association, had a full program for the many doctors' wives who were in Fort Worth and elected Mrs. R. C. Bellamy of Liberty as President-Elect. Mrs. Joseph H. McCracken, Jr., of Dallas succeeded Mrs. Mark H. Latimer of Houston as President.

Winners in the attendance contest, selected by lot from among those registered at any time during the annual session from the county societies showing the largest percentage attendance, follow: Dr. Herbert M. Alston, Gainesville (representing Cooke County, winner in the up to 25 membership classification with 100 per cent attendance), round trip for two to Mexico City via Compania Mexicana de Aviacion; Dr. Roland M. Shiflett, Jr., Cleburne (Johnson County, 26-50 classification, 77 per cent), round trip for two to Havana via Braniff International Airways; Dr. H. Glenn Walker, Midland (Andrews-Ector-Midland Counties, 51-100 classification, 26.4 per cent), set of luggage from Weaver's Prescription Pharmacy of Fort Worth; and Dr. Kenneth P. Wittstruck, Waco (McLennan County, 100 and over classification, 33.9 per cent), physician's bag from Wilson X-Ray and Surgical Company of Austin.

Those interested in comparative registration figures will want the breakdown on attendance at Fort Worth: members 1,643, special speakers (including 2 members) 31, visitors and students 195, exhibitors 192, Auxiliary 626, total 2,685. As mentioned previously, the total was a record, the nearest figures being 2,558 in Dallas in 1940 and 2,554 in Houston in 1953. However, the number of members present was exceeded in 1948 when there were 1,685 registered in Houston.

The swing toward increased attendance creates a problem of both meeting room and bedroom space which was acknowledged by the House of Delegates in giving the Board of Trustees, with the advice of the Council on Scientific Work, the right to change the place of the forthcoming annual session if it is deemed advisable because of available facilities. Tentatively, however, the 1956 session will be in Galveston from April 22 to 25, the 1957 session in Dallas from April 28 to May 1, and the 1958 session in Houston.

## A GIFT FOR THE ASSOCIATION

In 1951-1952, when the Texas Medical Association was still housed in a makeshift building but was looking forward to completion of its own headquarters within a few months, Mrs. O. W. Robinson of Paris was President of the Woman's Auxiliary. She was acutely aware of the lack of facilities then available and has rejoiced with everyone else over the modern building with its attractive auditorium-lounge, its kitchen, and its equipment for serving refreshments when meetings of the Association or Auxiliary are brought to Austin, as well as its library and office space.

To help complete the appointments of the Association building, Dr. and Mrs. Robinson have presented a handsome reproduction tray

in heavy silver plate, engraved on the under side: "The Texas Medical Ass'n. from Dr. & Mrs. O. W. Robinson, Paris, Texas." An oval measuring approximately 18 by 24 inches in the widest dimensions, the tray has ornate handles and legs and an intricately engraved design.

A gift so carefully chosen and one which will be enjoyed by Texas doctors and their wives down through the years deserves special recognition. It is from a chorus of 7,000 physicians and 5,000 Auxiliary members, then, that this thank-you comes for Dr. and Mrs. Robinson.

## PROGRESS REPORT—SALK VACCINE

When Dr. Thomas Francis, Jr., announced on April 12 that the Salk poliomyelitis vaccine had passed the extensive tests which were evaluated at the Poliomyelitis Vaccine Evaluation Center at the University of Michigan, he touched off a chain of events which had for months been carefully prepared for by doctors, manufacturers, educators, and health officers.

To insure that children in the high danger bracket would be the first to receive injections, the National Foundation for Infantile Paralysis ordered enough Salk vaccine to inoculate without charge all first and second grade children in the continental United States, Alaska, and Hawaii, and all children who were in the 1954 test group but failed to receive the vaccine. Only a small quantity, packaged in containers not useful for the mass school program, was available for private use.

The first plan was to give school children three initial injections; since then, Dr. Salk has recommended two injections, given two to four weeks apart, followed by a booster seven to twelve months later as the most effective schedule of inoculation.

Of the approximately nine million children who received the first injection, some subsequently developed poliomyelitis. These cases were centralized in a California area which has a high normal expectancy rate for the season, but they included other cases scattered throughout the country. The Salk vaccine was reported to be 60 to 90 per cent effective in last year's trial runs. Some authorities believe that since further tests and adjustments have been made, the figures may be higher this year.

Because many of the children on the West Coast who developed poliomyelitis were given vaccine from the Cutter Laboratories, Berkeley, the entire supply of Cutter vaccine was called in for testing. More recently, the government recommended temporary suspension of the program while experts reappraised manufacturing tests and processes in all laboratories.

Because of the highly limited supply of vaccine, the problem of distribution is receiving close attention. Mrs. Oveta Culp Hobby, Secretary of Health, Education, and Welfare, appointed a committee to study distribution and make recommendations to federal officials. Dr. Julian Price, a Trustee of the American Medical Association, was named a member of the committee, which discussed the voluntary system of allocation and various types of government control to assure just distribution. At this writing, no definite action on the problem of distribution has been taken.

By the end of July, officials report, enough Salk vaccine will be available to alleviate the fear of black market, but until that time, physicians in every county must work closely with health authorities to see that distribution is carefully planned and that priorities are established and followed.

Dr. E. S. McLarty, Galveston, introduced a resolution to this effect in the House of Delegates of the Texas Medical Association at the recent meeting in Fort Worth. The resolution, which was adopted, urged every county medical society to announce publicly a policy on priority which is considered best for its territory, and to seek to insure as fair and equitable distribution and use of the vaccine as is humanly possible.

# ORIGINAL ARTICLES

# DEVELOPMENTS IN THE CONTROL OF POLIOMYELITIS

S. EDWARD SULKIN, Ph.D., Dallas, Texas

I DO not think it is an exaggeration to say that the attention of the civilized world for the past two weeks has been focused on the evaluation of the greatest mass vaccine trial in medical history. The reports which have come from the Vaccine Evaluation Center in Ann Arbor, Mich., certainly have been favorable.

Because of the intensity of the accompanying fanfare, however, it has been difficult for the careful scientist to distinguish between public enthusiasm and cold facts—is the vaccine really 90 per cent effective? Will the use of the Salk vaccine eventually eradicate the poliomyelitis virus? Has the need for further research on poliomyelitis been eliminated? The answers to these questions require careful analysis of the evaluation reports and intensive study of the present status of the poliomyelitis problem. The answer to each of them is an obvious "no."

In order to arrive at an intelligent understanding of the present situation and the next most logical procedures, it might be of some value to review the steps which have led to the development of the present vaccine and a consideration of work still to be done in the future.

## STEPS IN DEVELOPMENT

The possibility of producing effective immunization against poliomyelitis has been considered seriously ever since the experimental transmission of the disease to animals was accomplished almost fifty years ago. For more than three decades investigations of the problem of immunization against poliomyelitis were hampered by sources of error due to lack of quantitative methods of assaying virus and antiserum, failure to recognize existence of different serologic types of virus, and lack of effective means for testing resistance to infection conferred by immunization procedures. New lines of research in recent years which have been moving forward on an unprecedented scale have resolved these problems to a great extent and have pointed the way to the development of successful immunization procedures.

The present hope for success that accompanies the renewed attempts at immunization against poliomyelitis is due in a large part to remarkable advances which have been made: advances in methodology, a better understanding of the pathogenesis of the disease itself, and the application of general immunologic concepts which have been well established for other viral diseases.

Much progress has been made in obtaining a clearer understanding of the etiology of poliomyelitis. Although it had been suggested several years ago that immunologic differences might exist between individual strains of poliomyelitis virus, it has been established only recently that at least three serologic types of this virus exist, designated the Brunhilde, Lansing, and Leon types, respectively. There is a possibility that a fourth type exists and that it is detectable only by the tissue culture method. Despite the fact that all three types produce identical clinical symptoms, infection with one will not produce antibodies effective against either of the other two. Each serologic type produces specific immunity or resistance to the disease when the patient recovers. A completely immunized person, then, must be protected against all types of poliomyelitis virus that may be introduced into his environment, and an effective vaccine must contain not only poliomyelitis viruses present in a particular area but those existing anywhere in the world.

Most important on the list of developments in recent years is the observation by Enders and his colleagues that the poliomyelitis viruses not only multiply in non-nervous tissue in the test tube but produce a characteristic cytopathogenic change by which the presence of virus can be readily recognized. This technique has served as an invaluable source of virus in a medium that is not as potentially harmful an inoculum as is nervous tissue.

The fundamental basis for active immunization against most infectious diseases is dependent first upon the behavior of the parasite in the tissues of the host and second upon the immunologic response of the host. Much recent data relate to the activity

*In the preparation of this discussion the author has drawn freely from the literature without making reference to the specific investigations.*

*From the Department of Microbiology, The University of Texas Southwestern Medical School.*

*Read before a General Meeting, Texas Medical Association, Annual Session, Fort Worth, April 27, 1955.*

of the poliomyelitis virus in both circumstances. Recently reported evidence that the virus may be present in the blood regularly, but briefly, early in the course of the disease and before the onset of paralysis is highly significant. These findings, together with a reappraisal of previous evidence, have led to a new concept of the pathogenesis of paralytic poliomyelitis. Formerly it appeared that the virus entered the alimentary tract through the mouth and that it made its way from this site to susceptible areas of the brain and spinal cord by way of peripheral nerve fibers. The new concept suggests that from the alimentary tract the virus is absorbed into the blood, where it initiates the rapid production of specific antibodies. From the blood stream the virus reaches the central nervous system where it attacks susceptible nerve cells by passing through the walls of blood vessels at an area of increased penetrability, and more importantly, this passage of virus into the central nervous system occurs during the brief period when not enough antibody is present to inactivate the virus in the blood. The newer concept would suggest, then, that there are two principal places in which virus may multiply in the host: (1) certain areas in the alimentary tract and (2) the nerve cells in the brain and spinal cord.

The sequence of events has an important bearing on the objective of immunization. Infection in the alimentary tract precedes that in the central nervous system, and in only few persons does involvement of the central nervous system occur. Although approximately 90 per cent of adults in this country have experienced a primary infection of the alimentary tract with the poliomyelitis virus without overt evidence of disease, less than 1 per cent of persons by the age of 20 years also experience the paralytic effects of subsequent infection of the central nervous system. If the presence of virus in the blood is an essential prerequisite to the paralytic disease, any procedure by which circulating antibodies may be introduced into the blood stream at the appropriate time would protect against paralysis by blocking the virus before it reaches the central nervous system. The possibility still exists, however, that virus also may reach the central nervous system by extension along nerve trunks. Moreover, it has been suggested that even if the latter is the case, there remains the possibility that involvement of the central nervous system may be prevented through a spilling-over of antibodies from the blood stream into nerve tissues. This is most likely to occur in persons in whom high antibody titers have developed following artificial immunization.

Although it is impossible to say with complete assurance whether the virus reaches the central nervous system of human beings by way of the blood stream or by way of the nerve fibers which supply the alimentary tract, in the experimental animal it can be shown that the virus is capable of penetrating the central nervous system by either route. Furthermore, virus can be recovered from the blood only during the preparalytic period, since by the time paralysis occurs the serum antibody reaches levels capable of neutralizing the virus in the blood stream. Virus responsible for the primary infection in the alimentary tract produces no known damage to the host and is excreted into throat secretions and in the feces. It may be excreted in the feces for several weeks after onset of the inapparent infection in the alimentary tract uninfluenced by the development of antibody in the blood serum. On the other hand, virus is present only for a few days in the throat secretions, a period of time roughly coinciding with the suspected contagious period. The presence of antibodies in the throat secretions may be responsible for shortening the period of time when virus is recoverable from the throat.

## IMMUNOLOGIC CONTROL

There are two main approaches to the immunologic control of poliomyelitis: (1) the use of virus variants which in small doses can infect and produce immunity without giving rise to paralysis or other symptoms of disease and (2) the inoculation of sufficiently large amounts of vaccines prepared with killed cultures of the three serologic types of virus to stimulate the development of immunity.

Extensive research on the first approach to vaccination against poliomyelitis, namely, the use of living avirulent variants of the virus, is desirable because experience with similar vaccines against smallpox and yellow fever indicate that a single inoculation may provide immunity for many years, if not for life. Recent experiments have shown that many strains vary in their virulence and tend to mutate like bacteria or other living things. Avirulent variants of all three types of poliomyelitis virus already have been demonstrated by several groups of investigators. In fact, there are now almost as many avirulent variants as there are workers in the field. Work toward the development of live virus vaccines is proceeding rapidly; and, although much remains to be learned about the best route of administration, the dosage, stability, and safety of this material, it is possible that this type of immunization ultimately may prove to be the more reliable and effective means of combating poliomyelitis.

It is the second approach to the problem, the use

of vaccine prepared with killed cultures, however, which has provided our first usable immunization medium. It has been shown that viruses inactivated by formaldehyde and other chemical and physical methods, although apparently noninfectious, still retain a considerable amount of antigenicity. Because of the outstanding success with vaccination against smallpox and yellow fever, it is frequently stated that only through the use of active virus can an effective and prolonged immunization be obtained. However, killed virus vaccines are not always inferior to live virus vaccines as stimulators of antibody production, especially when booster injections are employed. An inactivated vaccine in which representative strains of each of the three immunologic types are propagated in cultures of monkey tissue has been used in the extensive field trials. The virus is inactivated with formaldehyde. There can be little doubt that these trials have yielded encouraging results. I have had opportunity to study the Francis report since its release on April 12, and there can be no doubt that the use of the Salk vaccine will limit the occurrence of paralytic poliomyelitis.

One fact that definitely has been established by the vaccine trials is that the Salk killed poliomyelitis vaccine is safe. Assurance was gained before trials were undertaken that the vaccine would be subjected to careful requirements for safety before its use. These experiments were carried out carefully. Tests of the efficiency of different lots of vaccine used in the trial were not so accurate, the difficulty being the limitations of small numbers and standardization of production techniques. Nevertheless, it was apparent that the efficacy of different lots of vaccine with respect to the different immunologic types of virus varied tremendously. Standardization of the biologic is one very obvious need in future studies.

Analysis of the effectiveness of the vaccine is complicated by several factors. Foremost, of course, is the fact that, even though the immunization program was a huge one, the number of cases available for analysis was relatively small. Secondly, the tests were conducted on the basis of two study plans, an observed control plan whereby second grade children were inoculated and first and third graders were not inoculated but were kept under observation for the occurrence of poliomyelitis, and a placebo control study whereby children in the first, second, and third grades were combined, half receiving vaccine and the other half receiving a solution similar in appearance but having no influence on immunity to poliomyelitis.

Because of differences in classification and reporting of cases and numerous other variable factors, Dr. Thomas Francis, Jr., in his Summary Report of the Evaluation of the 1954 Field Trial of the Poliomyelitis Vaccine (April 12, 1955) states that "it was not possible to select a single value giving numerical expression in a complete sense to the effectiveness of vaccine as a total experience. If the results from the observed study areas are employed the vaccine could be considered to have been 60 to 80 per cent effective against paralytic poliomyelitis, 60 per cent against Type I poliomyelitis and 70 to 80 per cent effective against disease caused by Types II and III. There is, however, greater confidence in the results obtained from the strictly controlled and almost identical test populations of the placebo study areas. On this basis it may be suggested that vaccination was 80 to 90 per cent effective against paralytic poliomyelitis; that it was 60 to 70 per cent effective against disease caused by Type I virus and 90 per cent or more effective against that of Type II and Type III virus. The estimate would be more secure had larger number of cases been available." In this connection it is important to bear in mind that the type 1 poliomyelitis virus is the type which is largely responsible for epidemics of the disease, and it is, therefore, important to improve the efficiency of the type 1 component of the vaccine.

Although antibody responses to the type 1 virus are relatively low, it has been shown that sufficient antibody is produced with inactivated vaccines to prevent central nervous system invasion and paralysis, at least for a limited period of time. If this is actually the case, then virus exposure during the period of immunity would merely enhance the antibody level as a result of the alimentary infection. Furthermore, on the basis of data presented by Salk and his colleagues, even in the waning period of vaccine-induced immunity the antibody-forming mechanism is sufficiently sensitized to respond with a substantial booster effect to further virus exposure. This may occur even when the antibody titer is below the level of detection by present methods.

It would appear, then, that the critical issue on which the success of the present vaccine trial depends is whether the booster response under such conditions is rapid enough to prevent virus multiplying in the alimentary tract from overwhelming the body defenses and invading the central nervous system. If this is not the case, there is hope that better preparations of killed virus vaccine, when bolstered by booster injections, will be capable of producing a high enough level of immunity in the susceptible population. It is entirely possible that even relatively low levels of antibody, such as can be obtained with an inactivated vaccine, will consistently prevent paralytic poliomyelitis.

## FUTURE PROBLEMS

There is no doubt that with the favorable report a giant milestone on the long road of poliomyelitis research has been passed. Many problems, however, are still with us and remain to be solved. The duration of immunity conferred by the vaccine is as yet undetermined although there are indications that antibodies may persist for as long as twenty months after vaccination. Dr. Salk has recently recommended a modified schedule of inoculation. Obviously, this matter must be explored further before a precise procedure can be settled upon. It is conceivable that after an initial inoculation with an inactivated vaccine, a booster inoculation with a live vaccine eventually might be recommended.

Need for a procedure for successful shipping of monkey cells from India or the development of stable strains of normal cells to be used in the manufacture of vaccine are problems that are now occupying the attention of some research workers. A simple and easily preserved nutrient medium for propagating both the cells and the virus is an urgent need. It is not known whether vaccinated persons subsequently exposed to the virus will become symptomless carriers. It already has been shown that the presence of passive immunity resulting from the use of gamma globulin did not prevent the occurrence of natural infections. There still remains the possibility of a more effective or simpler vaccine being developed, perhaps through the use of attenuated strains of poliomyelitis virus. Studies along this line should not be abandoned.

The complete problem of poliomyelitis has not been solved. Much time will pass and many workers will contribute to the final solution of every facet of the problem. But let us not be discouraged by any of these or possibly other potential difficulties. We have or can develop techniques to meet present problems or new ones as they arise. With the background of accomplishment in research so forcefully evident we can go forward with confidence that a major victory has been won in the eventual conquest of poliomyelitis and that it should not be many years before paralytic poliomyelitis becomes a rare disease in vaccinated communities. Here in Texas we will have seen a disease make its first appearance, grow to epidemic proportions, and be controlled in the short space of two decades.

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# REFRACTORY CONGESTIVE HEART FAILURE IN THE ELDERLY PATIENT

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THE incidence of diseases of the heart and blood vessels is well known to increase with the age of the patient. Congestive heart failure is more frequently seen in the old than in the young, but its characteristics and treatment are fundamentally the same regardless of age. The age of the patient does not appear to be the main determinant in the prognosis. Age, however, may introduce certain differences in the etiology, complicating factors, and response of congestive heart failure.

In elderly persons, arteriosclerosis is more often an etiologic factor in congestive heart failure than in younger groups. An increasing incidence of arteriosclerotic heart disease was confirmed by us in a recent study[10] which showed such changes in the etiology of heart disease in the past twenty-five years. Recent analyses of life insurance statistics,[12] of congestive failure patients in England,[14] and of necropsies in Chile[6] led to similar conclusions regarding the importance of coronary atherosclerosis as the most common etiologic factor in the change of the cardiovascular system in the elderly.

It must be remembered, however, that the same causes may be present in the heart disease of the aged as in younger patients. Thus in addition to the frequent arteriosclerosis and hypertension, there may occur syphilitic, rheumatic, hyperthyroid, pulmonary, and even congenital heart disease. Acute rheumatic myocarditis recently has been again observed and reported in elderly persons.[5]

By refractory congestive heart failure is meant heart failure that is not responsive to the usual measures for its control. Its occurrence is of course not limited to the geriatric age group. However, oldsters are more prone to get into serious refractory states and are harder to bring out and keep out of electro-

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lyte imbalances. Its presence does not indicate that the situation is hopeless. It does obligate the physician to reinvestigate the clinical status of the patient along the following lines: (1) to make certain that the diagnosis is correct and complete, (2) to determine why the patient is not responding and whether the therapy was adequate as to drug dosage and dietary restriction, and (3) to note the effect of proper alteration trials in total therapy and management. If blood chemical studies are available, the total electrolyte pattern with blood chlorides and carbon dioxide combining power, sodium and potassium, nonprotein nitrogen, and serum albumin are the most accurate guides for the selection of reparatory measures. An acid urine with a hydrogen ion concentration of less than 7 suggests heavy chloride losses in the urine, and a high alkaline urine with a hydrogen ion concentration greater than 7 suggests a high sodium excretion.

## DIAGNOSIS

The diagnosis of congestive heart failure in its early stages is highly desirable as better results may be expected and the patient may be spared much discomfort by prompt institution of proper therapy. However, to diagnose congestive failure early, the physician must be on the alert for the condition. It is particularly difficult to recognize the early signs of decompensation in the elderly patient. Left ventricular failure should be suspected when there appear such symptoms as cough, shortness of breath on exertion, orthopnea, and particularly paroxysmal nocturnal dyspnea. Persistent rales at the lung bases may be the signs of pulmonary congestion or edema, instead of the erroneously diagnosed bronchitis. In addition to the signs of pulmonary congestion, there are often the additional findings of pulsus alternans or a gallop rhythm. Right ventricular failure usually appears after the left ventricle fails in diffuse coronary atheromatosis or hypertension. Congestive heart failure produces increased pressure in the systemic venous system, with distention of the neck veins, dependent edema, and a tender, enlarged liver. An elevated venous pressure is strongly suggestive, but not infallible, objective evidence, and the circulation time is usually prolonged. We have found the hepatojugular reflux of value in many borderline cases, especially if the venous pressure measured before and after manual compression of the liver shows a significant further rise.

The physician must resurvey critically the symptoms and signs and be certain that they are due solely to congestive heart failure rather than to other, contributing causes. In table 1 are listed some other possible causes for the various clinical findings that usually suggest congestive heart failure.

TABLE 1.—*Conditions at Times Mistaken for Congestive Heart Failure.*

1. Other causes of dyspnea.
   a. Pulmonary disease.
   b. Hyperthyroidism.
   c. Anemia.
   d. Obesity.
   e. Acidosis.
   f. Hyperventilation.
2. Other causes of rales.
   a. Bronchitis.
   b. Bronchiectasis.
   c. Atelectasis.
   d. Pneumonia.
   e. Asthma.
   f. Pulmonary fibrosis.
   g. Pulmonary infarction.
3. Other causes of dependent edema.
   a. Varicose veins.
   b. Phlebitis.
   c. Hypoproteinemia.
   d. Nephritis.
   e. Cirrhosis.
   f. Obstructing masses.
   g. Lymphedema.
4. Other causes of right upper quadrant tenderness.
   a. Hepatitis.
   b. Cirrhosis.
   c. Hepatic vein thrombosis.
   d. Cholecystitis.
   e. Peptic ulcer.
5. Other causes of neck vein distention.
   a. Superior vena caval obstruction.
   b. Constrictive pericarditis.
   c. Venous thrombosis.
   d. Tricuspid stenosis.

## CONTRIBUTING ETIOLOGIES

When a patient with congestive heart failure fails to respond to the usual therapeutic measures, the physician must be certain that the predisposing conditions have been adequately dealt with, that the precipitating causes have been controlled, and that perpetuating factors have not been overlooked. In table 2 are listed the more frequent complicating conditions which may perpetuate heart failure and which may be the cause of unresponsiveness to therapy. In particular, conditions such as masked hyperthyroidism, anemia, hypoproteinemia, or avitaminoses may be the cause of continuing failure. Prompt recognition and specific therapy of such complicating factors often result in a dramatic therapeutic response.

TABLE 2.—*Possible Contributing Etiologies in Refractory Congestive Heart Failure in the Aged.*

1. Anemia.
2. Avitaminoses—such as beriberi.
3. Nutritional factors—low proteins, obesity.
4. Thyroid malfunction—hyper or hypo.
5. Pulmonary factors—cor pulmonale.
6. Pericarditis.
7. Acute or subacute myocarditis.
8. Other factors—arteriovenous fistula, nephritis, etc.

## PRECIPITATING FACTORS

The immediate causes which have resulted in the development of heart failure should be searched for and properly eliminated. The importance of these

precipitating factors was emphasized by Sonnek,[14] who showed that in his geriatric patients with congestive failure who recovered, extrinsic precipitating factors were present in 84 per cent. Proper therapy of such conditions as listed in table 3 may be of

TABLE 3.—*Precipitating Factors in Refractory Heart Disease in the Aged.*

| | |
|---|---|
| 1. | Infections, such as pneumonia. |
| 2. | Physical activity—inadequate rest. |
| 3. | Myocardial infarction. |
| 4. | Cardiac arrhythmias. |
| 5. | Pulmonary embolism. |
| 6. | Drug toxicity—overdigitalization. |
| 7. | Dietary indiscretions. |
| 8. | Overloading with fluids and salt. |

greatest importance to the establishment of circulatory equilibrium.

The most common precipitating factor of congestive heart failure is infection, which increases metabolism, interferes with oxygenation if in the lungs, and has a toxic myocardial effect. The second most important precipitating cause is undue physical activity, even the effort of labored breathing or pulling on of a girdle. In such cases, adequate restriction of physical activity is of paramount value, with, if necessary, the use of sedatives and narcotics temporarily. Myocardial or pulmonary infarction may be the immediate cause of failure. Repeated pulmonary embolism, perhaps unsuspected, may be the reason why there is continuing dyspnea and edema. Arrhythmias with rapid ventricular rates may precipitate or perpetuate failure, and adequate slowing of ventricular action is essential to achieve compensation. Pleural effusion such as right hydrothorax, or ascites, or gaseous abdominal distention may perpetuate failure until removal.

## REVIEW OF THERAPEUTIC REGIMEN

The next step is for the physician critically to review each phase of therapy to make certain that his prescribed treatment is the best possible for the patient. He must determine whether his instructions are being followed carefully, for it may be the patient and not the congestive failure that is refractory. The elderly patient may be confused and not be able properly to carry out the treatment program. In such cases, some reliable member of the family must be instructed and made responsible for the carrying out in detail of the program of treatment.

Hospitalization is indicated if there is conspicuous and persisting failure. In the hospital, the patient's true status can be established and properly evaluated with routine clinical observations, and a rigid regimen can be maintained. The response can be carefully studied, using such helpful aids as daily determinations of fluid intake and output, venous pressure and circulation time, vital capacity, weight, and most significantly, electrolyte values.

In our series two features frequently not adequately carried out by the outpatient are the proper limitation of physical activity and the sharp or total restriction of sodium. We often have been faced with apparently refractory failure patients in the Heart Clinic, 90 per cent of whom increase their output of urine freely on exactly the same medications that they had been taking. In such instances the failure was not truly refractory, and the improved response was the result of the added rest and salt restriction.

The amount of digitalis necessary for complete digitalization varies from patient to patient, and either inadequate or toxic doses of digitalis may be the reason why the patient is not improving. If there is atrial fibrillation, the problem of control with digitalis is simple, for the aim is the reduction of the apical rate to from 60 to 70 per minute. If there is sinus rhythm, the determination of proper digitalis dosage may be more difficult. If the patient is not responding properly and there is no evidence of digitalis toxicity, it is usually desirable to increase the digitalis until there is reached either the point of therapeutic response or evidence of beginning toxicity. To determine this, Lanatoside C, 0.4 or 0.8 mg., may be given intravenously preferably under electrocardiographic control.

Some digitalis preparations appear to have an increased value over powdered leaf in such cases. Gitaligin was effective in our experience[7] in failure cases refractory to other digitalis preparations and has been reported as having a greater therapeutic range.[1] Acetyl-digitoxin, the newest of the purified glycosides, has the advantage over digitoxin of quicker dissipation. There are further advantages of acetyl-digitoxin in that toxic symptoms, which are usually gastrointestinal, tend to appear before the more dangerous cardiac arrhythmias develop.[4]

## DIURETICS

The judicious use of diuretics is one of the mainstays of therapy, and the organic mercurials are still the most reliable diuretic agents. We have preferred to use the newer compounds containing a sulfhydril group such as Thiomerin[9] and SU-1775 (Mersoban, Ciba) because of their decreased toxic effect on the heart muscle and the kidney tubular epithelium. These mercurial compounds are given subcutaneously in the smallest dose required to elicit a response (usually 1 to 2 cc.), but we have hesitated to administer them more often than each third day. It is important not to administer mercurials in active nephritis or uremia. Nevertheless, albumin, red cells, and azotemia as great as 100 mg. of nonprotein nitrogen can occur

in congestive heart failure, and the mercurials are not contraindicated in such cases.

We have encountered many patients with congestive failure, however, who were refractory to mercurial diuretics. Ammonium chloride in 6 to 8 Gm. doses per day for two to three days before a mercurial diuretic is given may reestablish responsiveness if the latter has been due to hypochloremia. Some of our clinic patients who have not done well because of infrequent visits have been taught to administer mercurials to themselves at home under periodic supervision. Most of them have responded dramatically, and complications have been nil. Aminophylline and Decholin at times have been of value in potentiating mercurial action.

Diamox, which inhibits carbonic anhydrase, has proved itself of considerable effectiveness as a well-tolerated oral nonmercurial diuretic, but at times its results are poor. We[8] have used it successfully as a substitute for mercurial diuretics. Some patients secrete urine better on Diamox than on mercurials, these most often having emphysema, cor pulmonale, or hypochloremic alkalosis. Other patients who have not done well on either diuretic alone have improved on the combination of Diamox orally in 500 mg. doses per day with subcutaneous mercurials each three days.

## SERUM ELECTROLYTES

Mercury apparently promotes diuresis by inhibiting the reabsorption of sodium and/or chloride, and also potassium, in the renal tubule. The loss of one of these may exceed the others, and often the mercurials seem to augment chloride diuresis more than sodium. With advancing years, the patient often becomes less able to compensate for therapy-induced electrolyte disturbances because of degenerative processes throughout the body, particularly in the kidney. The elderly patient, in addition, tolerates such disturbances poorly, and often relatively minor electrolyte shifts may produce marked symptoms.

A study of the blood electrolytes appears necessary in each case of congestive heart failure which is not responding to therapy, particularly if all the other causes have been eliminated. This is especially true if the patient has been maintained on a low sodium diet, with very frequent mercurial diuretics, full digitalization, and perhaps resins. An electrolyte imbalance particularly should be suspected when the urinary volume is depressed instead of increased by diuretics. Furthermore, increasing weakness, drowsiness, stupor, psychosis, anorexia, thirst, nausea, vomiting, congestion of liver, edema, weight gain, heart rate, and nonprotein nitrogen suggest significant electrolyte imbalance. In such instances, it is dangerous to continue to administer mercurial diuretics until the electrolyte pattern is clarified.

To evaluate fully the electrolyte picture in such cases, determination of the serum chloride, carbon dioxide, nonprotein nitrogen, sodium, and potassium are necessary. A rough estimate of the serum potassium can be made from the electrocardiogram, but for the sodium and accurate potassium determination, a flame photometer is necessary. The tabulated data on our patients upon which we base our opinions have been published in the *Revista Argentina de Cardiologia 20:*65-77 (March-April) 1953.

The practitioner may not have the laboratory facilities available to make these studies and may have to depend upon less definitive data. Considerable information may be elicited from determinations of the hydrogen ion concentration and chlorides in the urine obtained by using diuretics, for which simple tests are available.[3] If the urine chloride output is high and the hydrogen ion concentration shows an acid urine, the physician may reason that the patient is excreting an excess of chloride and is apt to develop a hypochloremic alkalosis. If the chloride excretion is high and the hydrogen ion concentration normal or alkaline, the patient must be excreting both chloride and sodium. If the chloride excretion is low and the hydrogen ion concentration alkaline, the patient is losing sodium.

TABLE 4.—*Electrolyte Disturbances in Congestive Heart Failure.*

| Factor | Low salt syndrome | Low Cl syndrome | Low Na syndrome | Low K syndrome | High Cl syndrome |
|---|---|---|---|---|---|
| Sodium | L | N | L | N | N |
| Potassium | N | N | N | L | N |
| Chloride | L | L | N | N or L | H |
| Carbon dioxide | L | H | L | N | L |
| Nonprotein nitrogen | N or H | N | N or H | N | N or H |

L = low; N = normal; H = high.

Table 4 is a summary of the values of the electrolytes in the various syndromes of electrolyte disturbance. Often there are multiple electrolyte alterations, and the patient thus will not necessarily fall into a single classification in the table.

The *low salt syndrome* is characterized by symptoms of dehydration and salt depletion, such as weakness, apathy, oliguria, thirst, nausea, vomiting, and muscle cramps. In this syndrome, the previous therapy has usually been too vigorous, with sharp salt restriction and frequent mercurial diuretics, or the patient may have a salt-losing nephritis. The values of the serum sodium and chloride are low, with low carbon dioxide and elevated nonprotein nitrogen levels. There may be an associated potassium deficit. The therapy consists of cessation of mercurial administration, and intravenous injection of 3 to 5 per

cent hypertonic saline solution, with restriction of the oral water intake. The amount required may be roughly estimated by calculation, assuming the water content to be 70 per cent of the body weight, and remembering that 100 cc. of 5 per cent saline contains 86 milliequivalents of sodium. However, only about half of the calculated deficit should be given slowly at first, with care not to overload the left ventricle.

The diagnosis of *hypochloremic alkalosis* or the *low chloride syndrome* cannot be distinguished from the low salt syndrome on clinical grounds as there are overlapping features, but the symptoms other than edema and oliguria are usually minimal. The relationship of low serum chloride to refractoriness has been emphasized by Keith and Whelan[11] and more recently by Stapleton and Harvey.[15] In the low chloride syndrome there develops a refractoriness to mercurials, and the evidences of congestive failure become more severe despite their use. The electrolyte studies show a low blood chloride level, an elevated carbon dioxide combining power, and normal sodium and usually normal nonprotein nitrogen levels. After the diagnosis is made, mercurials are stopped until electrolyte balance is restored. Ammonium chloride is usually given orally in doses of 6 to 8 Gm. daily. Dilute hydrochloric acid may be used to supplement ammonium chloride in 5 cc. doses four times daily, 20 cc. of 10 per cent solution being equivalent to 3 Gm. of ammonium chloride. If the patient cannot tolerate or absorb ammonium chloride by mouth, it can be given intravenously in 1 per cent solution up to 4 to 8 Gm. per day, but at a rate not exceeding 200 cc. per hour.

The *low sodium syndrome* is the converse of the low chloride syndrome and occurs particularly in patients with renal disease who have been treated by marked sodium restriction and frequent mercurial diuretics. Since ammonia is not available for excretion, there is a loss of fixed base as sodium. The symptoms are similar to those of the low salt syndrome, but more intense. The diagnosis is made by finding the serum sodium level well below the normal of 140 milliequivalents per liter, and as low as 120 milliequivalents per liter, and the carbon dioxide level lowered. The serum chloride may be normal, often because ammonium chloride has been given. The treatment consists in giving hypertonic saline solution or 1/6 molar sodium lactate, after which the patient may respond again to mercurials.

*Hypopotassemia* usually occurs in association with low sodium and/or low chloride levels but has been noted as an isolated finding, particularly if there is vomiting. The excretion of potassium is increased by mercurial action on the kidneys and also by the action of some of the digitalis glycosides. The diagnosis may be suspected by the characteristic prolonged Q-T interval and T wave change in the electrocardiogram, and the serum potassium value is decreased below the normal of 4 to 5 milliequivalents per liter. The condition can be corrected by giving 3 to 4 Gm. of potassium chloride or potassium citrate daily.

*Ammonium chloride acidosis occurs* in patients with congestive heart failure if there is renal damage and large doses of ammonium chloride have been administered continuously rather than intermittently for two to three days per week as we have recommended. Sleisenger and Freedberg[13] recently reported a series of such cases, in which 6 to 8 Gm. of ammonium chloride had been given for from seven to forty-five days. Clinical manifestations may include anorexia, nausea, vomiting, lassitude, stupor, areflexia, and Kussmaul breathing. There is an inability of the diseased kidneys to prevent continued loss of fixed base with chloride in the urine, and acidosis results. The laboratory studies show an elevated serum chloride level, marked acidosis, and elevated nonprotein nitrogen level. The treatment consists of stopping the ammonium chloride and administering 2 to 3 liters of 1/6 molar sodium lactate daily for one to three days. To prevent such complications, it is wise to limit ammonium chloride administration to three consecutive days per week, preferably the two days before and the day of the injection of the mercurial diuretic, particularly if noncontraindicating renal disease is suspected.

## ADDITIONAL MEASURES

There are additional measures which may be employed to advantage in the refractory case of congestive failure. Most of the elderly patients do have as a primary or contributing etiology coronary atherosclerosis with impaired myocardial circulation. In such patients, coronary vasodilators improve the blood supply to the heart muscle and thus improve the efficiency of the heart. We have preferred medications such as Peritrate, 10 to 20 mg., four times daily; niacin, 50 to 100 mg. four times daily; and aminophylline or Choledyl, 200 mg. three times daily, for this purpose.

The mechanical removal of troublesome accumulations of edema fluid in the serous cavities not only may be dramatic in relieving the respiratory distress of the patient, but may be followed by a brisk diuresis in the patient whose urinary output has been unsatisfactory. If there is marked elevation of systemic pressure, the careful administration of antihypertensive agents, especially Serpasil, with care not to drop the pressure to excessively low values will help to relieve the load on the left ventricle.

The physician must remember to try to compensate for the loss of potassium from the myocardium in heart failure and also that which is incident to digitalization, which apparently increases the potassium excretion. It thus would appear to be good practice to augment the potassium by giving potassium chloride by mouth if there is no evidence of renal insufficiency.

The carbohydrate content of the diet should be high, as glucose is a good source of energy for the myocardium. Proteins should be given in amounts of 1.5 to 2.0 Gm. per kilogram of body weight to prevent hypoproteinemia. Antithyroid preparations such as radioactive iodine have been used in euthyroid patients with refractory congestive heart failure with apparently favorable results.

## THE FUTURE

Notable advances have been made in the past thirty years in the treatment of congestive heart failure. Patients now can be maintained in comfort for years whereas in the early part of the present century they would have been lost in a matter of weeks or months. Yet the problem has not been solved. Patients continue to die from congestive heart failure. All too often, the electrolyte disturbance underlying the refractory case can be corrected only after great effort, but the restored pattern is fragile and one mercurial injection may again precipitate the whole imbalance. Furthermore, the electrolyte disturbance rather than being secondary to therapy may be the electrolyte disintegration which is also observed in death from other causes.

Our future advances in the therapy of congestive heart failure probably will come from a better knowledge of the nature of the process and from fundamental investigation regarding the metabolism of heart muscle. Studies of the actin and the myosin, of adenosine triphosphate, and of the phosphocreatine metabolism all are necessary basic investigations. Elucidation of the chemical reaction of the myocardium in contraction may help eventually to strike at the problem of myocardial weakness at its very source. Perhaps through the results of some of these studies will be found the key which will unlock the basic mysteries of myocardial metabolism and the true underlying causes of myocardial failure.

## SUMMARY

Refractory congestive heart failure is managed similarly in all age groups, but in the elderly, coronary atheromatosis is a more frequent etiologic factor.

Proper therapy of precipitating, contributing, and perpetuating causes is essential in the refractory patient with congestive heart failure.

A review of the therapeutic regimen may bring about changes in therapy with gratifying results. Proper attention must be paid to the restriction of sodium, limitation of activity, adequate digitalization, and optimum use of diuretics.

The geriatric patient is more susceptible to electrolyte complications and is less able to withstand electrolyte imbalances and maintain reparations by solutions than the young patient. Adequate electrolyte studies are necessary in the refractory case, although determinations of hydrogen ion concentration and chloride in the urine may be of much value. Proper correction of serum electrolytes may again make the failure responsive.

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### New Measles Diagnosis

A simple laboratory test has been found helpful in diagnosing measles before the tell-tale rash appears, two New York physicians report. Patients in the stage of measles before rash appears develop an unusual type of cell in the membranes of the nose and throat. Samples of such cells were found regularly in 10 patients from five days before rash to the day it appeared. Drs. Victor Tompkins and John C. Macaulay, Albany, N. Y., of the New York State Health Department and the Albany Medical College described the test in the February issue of *The Journal of the American Medical Association.*

# INFECTIOUS HEPATITIS

## A Summary of Recent Information

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ALTHOUGH as recently as 1940 little was known about the disease now known as infectious hepatitis, outbreaks of the disease are reported to have occurred in troops during the Napoleonic Wars, the American Civil War, the Spanish-American War, and both World Wars. Indeed, it has been stated that infectious hepatitis was the greatest cause of disabling illness in the United States forces in the Mediterranean Theater during World War II. In times past, names applied to this disease have included infective hepatitis, epidemic catarrhal jaundice, nonspirochetal infectious jaundice, and campaign jaundice.

Infectious hepatitis is now known to be caused by a filtrable virus or group of viruses, although specific strains thus far have not been identified. The classification of infectious hepatitis as virus IH hepatitis as opposed to virus SH hepatitis (homologous serum hepatitis) has been suggested by Neefe.[14]

The virus of infectious hepatitis is difficult to destroy, being able to withstand heating at 56 C. for thirty minutes and to resist the usual concentration of chlorine added to drinking water. It has been demonstrated that 1.0 parts per million of residual free chlorine will destroy the virus in thirty minutes. With previous coagulation and settling of organic material in water, however, the virus apparently can be destroyed by 0.5 parts per million of residual free chlorine in thirty minutes. A major obstacle in working with the virus to determine its properties has been the lack of a susceptible laboratory animal. The only available method of investigation has been the use of volunteer human subjects.

The incubation period of infectious hepatitis is variable but is thought to be in the neighborhood of fifteen to fifty days with an average of about twenty-five days. Clinical hepatitis has appeared in volunteer human subjects at an interval of from seventeen to thirty-four days following the administration of infective material.

Diagnosis of infectious hepatitis may be difficult during the preicteric stage and often is simplified greatly by the appearance of clinical jaundice. However, since a large number of cases never become icteric, the diagnosis often may have to be made without the aid of such a diagnostic sign. Estimates of the frequency of clinical icterus in adults have ranged from 30 to 80 per cent with a considerably smaller percentage of children becoming icteric. During epidemic conditions the clinical syndrome may be recognized in a large percentage of cases prior to the onset of icterus. As there is no specific diagnostic test for infectious hepatitis, the diagnosis must depend on clinical and epidemiologic grounds with some assistance from nonspecific but usually consistent laboratory procedures.

The disease typically begins with the onset of a low-grade fever of 99 to 101 F., chilliness, generalized aches and pains, symptoms of an infection of the upper respiratory tract, and often nausea and vomiting. At this stage, the liver usually is not enlarged although there may be some tenderness in the epigastrium or under the right costal margin. The initial stage usually lasts three or four days and is followed by a one to four day relatively symptom-free period. This period of apparent improvement is in turn followed by the rather sudden onset of anorexia, nausea, vomiting, and the clinical symptoms of hepatic dysfunction with liver enlargement, icterus, clay-colored stools, and bile pigments in the urine. To be sure, all cases do not follow this pattern or display all symptoms. The disease often fails to display such clear-cut stages in younger patients.

In general, mortality appears to be low, reported as less than 0.5 per cent. The severity of the disease appears, in general, to have a reciprocal relationship to age, with severe illness and jaundice being relatively less common in younger age groups. Other factors appearing to influence the severity of a given case include (1) the general health of the patient and the presence or absence of preexisting liver disease, (2) adequacy of rest and nutrition during the acute illness, (3) intercurrent infection, (4) excessive alcohol intake, and (5) possibly pregnancy. Clinical relapses do occur but have been reported as less than 5 per cent in many surveys.

During the preicteric period of infectious hepatitis, other diseases to be considered in the differential diagnosis include upper respiratory infection, atypical pneumonia, infectious mononucleosis, and influenza. With the appearance of icterus, the following diseases should be ruled out: jaundice associated with such infections as cholangitis, leptospirosis, yellow fever, and infectious mononucleosis; hemolytic icterus, either congenital or acquired; hepatocellular jaundice

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associated with cirrhosis, hepatotoxic chemical agents, and carcinoma; and obstructive jaundice associated with either biliary calculus or neoplasm.

The disease appears to have no geographic boundaries and occurs throughout the world in all climates. It may occur as sporadic cases or in epidemic form at any time of the year, although a definite seasonal trend is noted. Cases usually increase in number during late fall and early winter, then decrease to reach a low point during late spring and summer. Epidemics usually are noted to occur under conditions of crowding and lack of good sanitation and personal hygiene such as may be present in wartime and in camps, in institutions, and within certain family groups.

Gauld[5] stated that the typical epidemic is not explosive in nature but follows a slow, leisurely course through a given group of persons taking three to three and one-half months to complete its course. Several explosive type epidemics of infectious hepatitis that were traced to infection through some common vehicle, usually drinking water, also have been reported.

Lacking any known host other than man, evidence points to transmission by clinical or subclinical human cases of the disease. The general picture in most epidemics reveals some form of person-to-person dissemination as the most important mode of transfer with both epidemiologic and experimental evidence supporting the intestinal-oral route. Respiratory transmission has been postulated, but attempts to recover virus from nasopharyngeal secretions have been repeatedly unsuccessful.

Experimental work has shown conclusively that the virus of infectious hepatitis is present in the stools of patients whether they have been infected by the oral or the parenteral route. The virus has been recovered from the blood and feces of patients with infectious hepatitis, and human volunteers repeatedly have been infected by both oral and parenteral administration of infective material. Available evidence leaves little doubt that the virus is present in the stool of patients and that the mucosa of the gastrointestinal tract may serve as a portal of entry for the virus. An additional point of importance is that although the usual mode of entry of the virus may be through the gastrointestinal tract, it has been demonstrated that the virus also may be transmitted by parenteral injection. Thus, infectious hepatitis as well as serum hepatitis may be spread by the use of contaminated blood or blood products or by the use of improperly sterilized instruments.

No particular racial susceptibility has been demonstrated for the disease, and it appears that both sexes are equally susceptible. Age appears to be an important factor in susceptibility with the age group under 15 years contributing the major number of cases in most epidemics. Army experiences in the Mediterranean Theater also have shown a decreased incidence in men over the age of 30. The factor of age suggests that immunity has been gained by the older population. Experimental data indicate that an attack of infectious hepatitis is followed by a relatively permanent resistance to reinfection with the same virus. This evidence of acquired immunity has been the basis of the use of gamma globulin as a prophylactic measure, and evidence now indicates that gamma globulin does offer relatively effective protection if given more than a week before the expected date of onset. Furthermore, it has been demonstrated that the passive immunity may be converted gradually to an active immunity if the person continues to be exposed to the disease, probably through the development of a subclinical case of infectious hepatitis.

Second attacks of infectious hepatitis have been reported with frequencies estimated at 2 to 5 per cent in areas where exposure was believed to be great.

The chief consideration in the prevention of infectious hepatitis is the prevention of direct or indirect transmission of the virus from the feces of infected persons to other persons by means of good community sanitation and personal hygiene as employed in the prevention of other enteric infections. The exact period during which the feces continue to contain virus is not definitely established, but it seems reasonable to consider that the period would exist at least for one month or until signs of acute illness are no longer present. Passive immunization of close contacts with gamma globulin is possible using 0.02 cc. per pound of body weight, although some authors have recently recommended as little as 0.01 cc. per pound of body weight.

Although virus SH hepatitis or homologous serum jaundice has been regarded by some to represent the accidental production of infectious hepatitis, experimental evidence points to the fact that the two diseases are caused by different viruses. Attempts to demonstrate the SH virus in the feces of patients have been unsuccessful, but the virus has been demonstrated in the blood of patients during the incubation period and in experimentally inoculated volunteers for as long as eighty-seven days before the onset of icterus. Blood, plasma, or serum from established cases of serum hepatitis inoculated in human subjects has been shown to produce hepatitis in from 40 to 160 days.

It has been observed by Stokes[21] that gamma globulin does not protect against or modify serum hepatitis although it occasionally may produce some pro-

longation of the incubation period. After recovery from virus SH hepatitis, volunteers were susceptible and possibly more susceptible than the normal person to virus IH hepatitis. Attempts to produce disease by the oral administration of the SH hepatitis virus have been unsuccessful. Thus, the available data suggest a definite difference in the two similar diseases —both in antigenic properties and in the mode of transmission.

As noted previously, a great deal of information about infectious hepatitis has been accumulated in recent years, but many aspects of the disease require further study. More direct knowledge of the epidemiology and better control measures certainly are needed. However, with the means presently at hand, it seems probable that many cases may be prevented in the future.

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## No Neurologic Damage After Spinal Anesthesias

Results of a study of 8,460 patients given 10,098 spinal anesthetics at one hospital have revealed no instances of major neurologic damage after postoperative follow-ups of as much as five years, it is reported in the *Journal of the American Medical Association* for December 18, 1954.

The study was made by Drs. Robert D. Dripps and Leroy D. Vandam at the Hospital of the University of Pennsylvania between 1948 and 1951. Based on their findings and other medical data, they term as "unjustified" the tendency "to assume a cause and effect relationship between spinal anesthesia and a variety of complaints, sometimes appearing years after the anesthesia.

"Our experience indicates that the mortality rate following spinal anesthesia is lower than that recorded after general anesthesia in comparable patients undergoing comparable types of operation."

Of the 10,098 spinal anesthetics administered, almost 75 per cent, 7,127, consisted of Pontocaine. The second most frequently employed agent, procaine, was given 1,399 times. A check made at least six months following 8,987 "spinals" disclosed one instance of incapacitating neurologic disease. This later proved to be due to a spinal cord condition "that prior to spinal anesthesia had caused no recognizable symptom," the authors stated, adding the patient subsequently recovered completely.

Numerically, headache was the most common postoperative complaint. Others included backache; pain and numbness in the buttocks, thighs, legs, and feet; and occasional weakness in leg muscles. The majority of complaints disappeared with the passage of time, the authors reported.

They attribute the absence of serious neurologic damage to the precautions observed in the study. These included careful selection of patients for a "spinal"; use of a meticulous technique that, long before the anesthetic is given, involves preparation in the way of cleansing and sterilizing equipment; choice of reputable pharmacologic products; use of safe concentrations as the anesthetic mixture; and careful technique in performing lumbar puncture and injecting the anesthetic.

### Cancer Research Grants

Three members of the Texas Medical Association are among the Texas scientists receiving research grants from the American Cancer Society. They are Drs. Vincent P. Collins, R. K. Loeffler, and Charles L. Spurr, Houston. Texas scientists received a total of $103,694 for cancer research projects.

# RECENT PROGRESS IN BILIARY TRACT SURGERY

FRANK GLENN, M. D., New York, New York

THERE has been increasing clarification in our thinking as concerns biliary tract disease and its management over the past quarter of a century. It has been generally established that gallstones are associated with the majority of disorders of the biliary tract. As a result, in the consideration of biliary tract disease one may more clearly present a comprehensive picture of this clinical problem by primarily following the natural course of gallstones and noting specifically those instances when calculi are not involved. Gallstones so often precede acute cholecystitis, common duct stone, liver damage, and even carcinoma of the gallbladder that in a sense they may be considered sequelae or complications of cholelithiasis. This concept has had an influence on the advancements in the management of patients with biliary tract disease.

It is my purpose to discuss briefly the progress we have observed on the surgical pavilions of The New York Hospital-Cornell Medical Center over the past two decades, during which time almost 3,800 patients were treated surgically.

## PATHOLOGY, SYMPTOMS, AND HISTORY

There has evolved slowly a clearer understanding of the pathologic changes and their relationship to the symptoms they cause. Although most biliary tract disease is associated with stones, noncalcareous conditions are recognized particularly among those with acute cholecystitis. We are still without sufficient knowledge about the etiology of gallstones to be able to prevent their formation; however, the natural history of biliary tract disease as associated with them has been clarified in particular in young women recently pregnant. This has corrected somewhat the tendency fostered by many textbooks of limiting their consideration to the "fat, fair, and forty." Also there has developed an appreciation of a difference in gallstone disease between that appearing in young women recently pregnant and older men and women not previously pregnant, as well as those with blood dyscrasias. The term "acute cholecystitis" has come to denote a phase of biliary tract disease that may be accompanied by complications of a serious nature. From studies of postmortem material it has been demonstrated that those with gallstones have an increased incidence of associated systemic changes such as obesity, hypertension, arteriosclerosis, diabetes, and renal impairment.

## DIAGNOSIS

Critical evaluation of information upon which a diagnosis of biliary tract disease is made has become more common but should be further extended. A clinical story compatible with gallstones supported by cholecystographic evidence too often has obscured the true cause of the patient's complaints. Improvement in differential diagnoses has developed not only because of better x-ray and laboratory procedures for evaluation of other nearby structures but also because of increased clinical experience, including follow-up studies.

After the introduction of cholecystography in 1924 there was a tendency, not yet completely overcome, to ascribe almost any upper abdominal complaint to visualize gallstones or a nonvisualized gallbladder. The careful keeping of records over the years and their periodic evaluation at intervals in various clinics has done much to keep the surgeons alert to the common mistakes. As a result diagnosis has become more accurate. Those conditions found most closely to simulate biliary tract disease in order of occurrence have been (1) peptic ulcer, (2) pancreatitis, (3) renal and perirenal disease including tumors of the adrenal gland (carcinoma of adrenal, pheochromocytoma), (4) coronary heart disease, (5) carcinoma of the hepatic flexure of the colon, and (6) acute appendicitis.

For the most part biliary tract disease is associated with stone formation. Acute inflammatory conditions do occur in the gallbladder without stones and usually are associated with some systemic disease. Then there are the rarer chronic inflammatory processes such as benign stenosing fibrosis of the ductal system and nonmalignant neoplastic lesions that may arise any place within the biliary system. Again careful clinical observations and follow-up studies have demonstrated that the removal of the gallbladder without gallstones rarely relieves symptoms in chronic disease. (In acute cholecystitis this does not apply.) We have become so impressed with these results that cholecystectomy is infrequently done and then only after consultation and deliberation if stones are not demonstrated at operation.

## PREOPERATIVE PREPARATION

We are now considering for operation patients in a wider age range than we did twenty years ago.

*From the Department of Surgery, The New York Hospital—Cornell Medical Center.*

*Read before the International Medical Assembly of Southwest Texas, San Antonio, January, 1955.*

First, are the younger persons. We recognize gallbladder disease in children although it is rare. Far more important are the young women recently pregnant who have gallstones and the symptoms they give rise to. Once a diagnosis is established surgery is recommended and usually accepted regardless of whether the cholecystitis is in an acute or a chronic phase. Formerly these patients being young and vigorous were encouraged to tolerate their symptoms and avoid operation. These patients in our community are well informed concerning gallstones and come to the hospital seeking relief.

There has been a steady increase in the proportion of patients 65 years and over. No doubt this parallels a comparable increase in the overall population. All patients before undergoing surgery for biliary tract disease are more carefully evaluated. Diagnosis and differential diagnosis have been emphasized already. Immediately our concern is extended to the functional capacity of the liver and its probable reserve under stress of operation or subsequent complications. Diseases of the liver such as cirrhosis and accompanying portal hypertension are far better understood and managed. Nutritional disturbances manifested by overweight or symptoms of starvation are now directly attacked and in part corrected before surgical procedures are undertaken. Roentgen-ray examination and electrocardiograms supplementing clinical evaluation of the cardiorespiratory systems provide the surgeon with information that aids in the selection of the optimum type of anesthesia. Careful preoperative evaluation is followed by steps to correct deliberately any condition that is amenable so that the patient is in as good condition as possible before the burden of an operation is added. To mention a few of these such as hypoproteinemia, electrolyte deficiencies, diabetes, and dehydration is to indicate that we are better able to correct them than formerly.

## HEMORRHAGIC TENDENCY

One of the most important contributions to the management of the jaundiced patient with an increased hemorrhagic tendency has been the introduction of vitamin K. Measured in terms of prothrombin time the bleeding tendency is recognized in degree. Where the liver parenchyma has not been destroyed the administration of vitamin K corrects the bleeding tendency. The mortality following surgical operations upon jaundiced patients was formerly very high. The hemorrhagic tendency due to vitamin K deficiency in the jaundiced patient has been overcome, and we have not lost a patient because of bleeding since the vitamin was first made available in 1937.

## CHEMOTHERAPY

The role of chemotherapy is rather difficult to evaluate properly. That it has contributed to the better control of infections of the biliary system, particularly cholangitis, there is no doubt. We have examples of patients who have had severe infections who were markedly and quickly improved. One woman with common duct stones and a cholangitis treated by chemotherapy prior to operation developed miliary liver abscesses with organisms resistant to the antibiotics she was receiving. Selection of suitable antibiotics was followed by recovery. In my opinion she surely would have died without this therapy. However, over the past five years we have come to use less chemotherapy before and after operation. There have been very few complications and not a single fatality due to infection. The indications for chemotherapy have become more clear-cut. Chemotherapy is not used routinely as a prophylactic measure.

## ANESTHESIA

Two evident advances have been made in regard to anesthesia. The first deals with the selection of the anesthetic agent. Specific consideration is given to the status of the liver and the cardiovascular system. Impaired liver function requires a minimal amount of the least toxic agent available. The damaged liver may be unable to participate in the elimination of the agent. The second and perhaps more important aspect of anesthesia is its administration. The art of providing adequate relaxation to facilitate the operation and at the same time using a minimal amount of anesthetic agent together with a respiratory atmosphere high in oxygen has been developed to a degree unanticipated twenty years ago. Postoperative pulmonary complications have been greatly reduced by preventing aspiration of gastrointestinal contents and the removal of excess bronchial secretions by suction during the operation. These have been made possible by (1) evacuating gastric contents before induction and during the procedure by means of an indwelling nasogastric tube and (2) the employment of the intratracheal tube in all patients being operated upon under general anesthesia.

These measures followed by early mobilization, that is, having the patient stand and then walk about his bed as soon as he is completely conscious after the operation, are in large part responsible for reducing the incidence of fatal pulmonary complications subsequent to operations on the biliary tract.

## OPERATIVE PROCEDURES

More adequate exposure for the procedure to be undertaken has been obtained by selecting the incisional approach according to the habitus of the patient. The longitudinal incision is used for those

of asthenic build, reflecting and not splitting the right rectus muscle. For those of a more sthenic stature a subcostal incision with transection of the right rectus muscle is employed.

A methodical survey of the abdominal contents before approaching the biliary tract unless contraindicated has on occasions revealed an unsuspected finding.

In evaluating the biliary tract we have become more concerned than formerly with the condition of the pancreas and the area of the duodenum in the region of the ampulla of Vater. Cancer in the older age group, and more of our patients are now in this category, is being looked for.

Since 1932 we have stated on various occasions that in the performing of a cholecystectomy we believed it made little difference whether the gallbladder was removed from the fundus down toward the cystic duct or from the cystic duct toward the fundus. In instructing the resident staff we expressed the opinion that whichever method seemed the easier for the operator should determine the approach. We are now departing from this point of view, and it is recommended that the first step in a cholecystectomy be the identification and dissection of the cystic duct to demonstrate its junction with the common duct. A ligature is then placed about it for identification and traction. Next the cystic artery is dissected free to its origin, usually from the right hepatic artery, and then temporarily occluded by ligature near its junction with the wall of the gallbladder. The gallbladder is then dissected from the fundus downward, the operator exercising great care to stay within the capsule of the gallbladder bed and not to injure the liver. If scarring is present it is considered preferable to open into the gallbladder rather than to penetrate the capsule or injure the liver. The temporary occlusion of the cystic artery renders this a relatively bloodless procedure. As the cystic artery is encountered it is secured and permanently ligated.

This method of cholecystectomy is advocated because we believe that injury to the common duct will be much less likely. Over the period of this review I have talked with several surgeons who have referred cases to us because of common duct injury during cholecystectomy and were willing to describe in detail their operation. In each instance they had sought to remove the gallbladder from below toward the fundus. They had not satisfactorily dissected the two vital structures, the cystic duct and the cystic artery, separately and demonstrated the junction of the cystic and common duct beyond all question of a doubt before they divided the duct.

Reperitonealization of the gallbladder bed is done only when this can be accomplished easily without creating a dead space. If the gallbladder has been removed without damage to the liver and vessels and accessory ducts ligated as encountered, drainage from this surface is of no significance. Bringing the peritoneal margins over an injured surface may not prevent its further weeping.

We advocate and practice the placing of a drain extending from the region of the cystic duct remnant along the course of the gallbladder bed and through a stab wound in the flank, not through the operative wound.

## COMMON DUCT OBSTRUCTION

Our stated indications for exploration of the common duct remain unchanged. These are (1) palpable stone within the common duct, (2) thickened wall of the common duct, (3) dilated common duct, (4) dilated cystic duct, (5) presence or history of jaundice, (6) frequently when there is thickening or enduration of the head of the pancreas, and (7) frequently when patients are over 65 years of age.

*Cholangiography.*—Unless there is a contraindication such as the condition of a patient being precarious or an existent cholangitis, cholangiography is recommended whenever common duct exploration is decided upon. Both procedures add considerable to the operation; they should not be entered into lightly. They should be done with great care when undertaken. In performing cholangiography at operation two objectives are fundamental. The first is the complete filling of all the patent ductal system, and second, the securing of roentgenograms that visualize the ductal system without overlay of the opaque material that may obscure any part. To accomplish this, oblique views that demonstrate the lower end of the common duct in its entirety separately from the duodenum are essential.

Exploration of the common duct was done in the early part of the past twenty year period in less than 10 per cent of patients. This rate has been increasing and continues to increase so that now it approaches 17 to 18 per cent. Our indications remain the same, but the mean age of the patients has increased. Stones are recovered in about 50 per cent of those subjected to exploratory surgery.

## POSTOPERATIVE MANAGEMENT

The immediate postoperative management has included early mobilization over the past ten years. This, together with a decrease in postoperative complications, has enabled us to reduce the period of hospitalization from an average of approximately fourteen to nine days. Better anesthesia and early mobili-

zation have been factors in the reduction of postoperative pulmonary complications.

Diet after operation has been an object of special study that has resulted in changes. Food by mouth is begun as soon after operation as tolerated. On discharge patients are instructed not to eat large amounts and not to allow themselves to become over-hungry. Small meals at short intervals have been successful in keeping patients comfortable. No special restrictions are placed upon fat intake. A well-balanced diet that maintains the patient's normal weight is recommended.

Symptoms that are similar to those prior to operation or suggestive of biliary tract disturbances are an indication for intensive reevaluation. There are a group of patients who following surgery upon the biliary tract have a persistence or a recurrence of symptoms. For the most part these symptoms have been right upper quadrant or midepigastric pain, nausea, vomiting and/or intolerance to fatty food or large meals. Some of these patients have had varying degrees of jaundice. Intravenous cholangiography provides an additional means of demonstrating previously overlooked stones in the common duct, remnants of the cystic duct, and other abnormalities. This, together with a complete reevaluation seeking evidence of other conditions such as duodenal ulcer, hiatus hernia, pancreatitis, and renal disease, has reduced the number of "post-cholecystectomy syndrome" patients.

## "SILENT STONES"

It long has been known that many persons at postmortem are found to have stones in the biliary tract with no available history that they had produced symptoms during life. This gave rise to the term "silent gallstones." During the past twenty years a greater proportion of our population, particularly those over 50, periodically undergo complete clinical evaluation, including cholecystography. Unsuspected cholelithiasis is thus demonstrated. Many surgeons are asked to see these patients in consultation. Our attitude at present is to recommend cholecystectomy unless there is some contraindication. One cannot predict the future course of the "silent stones"; they may cause an obstructive acute cholecystitis of a rapidly culminating type in the older age especially. The surgeon should consider the risk of operation in the light of possible complications that may occur in patients with biliary calculi including carcinoma.

The age factor in the surgical treatment of biliary tract disease in our experience at The New York Hospital-Cornell Medical Center is considered to be of great importance. Among patients less than 50 years of age the rate of fatal complications in nonmalignant disease of the biliary tract has been 0.65 per cent. Among patients 50 to 64 years this rate is 2.5 per cent; and among those patients more than 65 years of age it is increased to 6.7 per cent. For elective surgical procedures the difference in mortality is not significant. The high mortality rate among aged patients occurs following emergency surgery. Thus the complications of acute cholecystitis in the aged patient with degenerative disease are the chief factor in the operative risk. Surgery should be done in older patients with acute cholecystitis; but they merit the most meticulous care in surgical management. This includes operation as early as possible in an acute attack before complications such as perforation, stone obstruction, and jaundice appear. Our experience appears to justify the policy of regarding all calculous cholecystitis, whether symptomatic or not, as a potential hazard and an indication for elective cholecystectomy. Unique aspects of biliary tract disease in the aged include (1) an increased incidence of acute cholecystitis; (2) an increased incidence of choledocholithiasis with larger and more numerous calculi; (3) the occurrence of acute processes in the biliary tract with minimal signs and symptoms; and (4) the increased incidence of associated biliary tract neoplasia.

## SUMMARY

There has been considerable progress made in the management of biliary tract disease during the past twenty years. There are several reasons for this. First, there is a better understanding of the pathologic changes and their relation to the symptoms they give rise to. Second, accuracy of diagnosis has been improved and more attention has come to be given to those conditions that are commonly confused with biliary tract disease. Third, the operative treatment has become much safer and more thorough so that surgical treatment has to a large extent replaced medical management.

Contributing to advancement of surgical treatment has been (1) the control of the hemorrhagic tendency in the jaundiced patient with vitamin K, (2) chemotherapy to control infection, (3) critical selection and better administration of anesthesia, and (4) a greater awareness of anatomic variations of the structures encountered during a cholecystectomy and postoperative measures that provide a minimal amount of immobilization and invalidism.

The concept that cholecystectomy should be done for cholelithiasis unless there is some contraindication soon after the diagnosis has been established seems sound, even if the stones may not be producing symptoms because gallstones so often precede acute cholecystitis, common duct stone, liver damage, and even carcinoma of the gallbladder.

# TUMOR SEMINAR

*Conducted by*

*FRANK W. FOOTE, JR., New York, New York*

THE tenth annual tumor seminar sponsored by the San Antonio Society of Pathologists at Brooke Army Hospital, Fort Sam Houston, Texas, November 7, 1953, was opened by Col. Carl J. Lind, Jr., who introduced in turn the commanding generals of Brooke Army Medical Center and Brooke Army Hospital.

Credit for the development of the current seminar was given to Major Harry Sproat, MC, as coordinator, Mr. Henry Morasco as photographer, Capt. Francis J. Sullivan as administrator in charge of general arrangements, and Mesdames H. A. Watson and A. C. Krukowski for the secretarial work. Dr. A. O. Severance of San Antonio, secretary-treasurer, was concerned with the correspondence and contact of all pathologists who attended. Dr. B. F. Stout of San Antonio was honored again as the originator and founder of the San Antonio Society.

Generous grants from the South Central Regional Committee, College of American Pathologists and the American Cancer Society, Texas Division assisted in the support of this seminar.

Dr. Frank W. Foote, Jr., of Memorial Hospital, New York City, conducted the seminar.

## CASE 1

*Diagnosis.*—Adenoid cystic carcinoma.

*Contributor.*—Col. C. J. Farinacci, Fourth Army Area Medical Laboratory, Fort Sam Houston.

*Specimen.*—Mass from palate.

*History.*—This white woman, aged 29, gave the history that for one year she had pain in the left infraorbital region. She noticed bulging of the left palate one and one-half months before biopsy. At biopsy there was a large tumor mass protruding from the palate.

*Submitted Diagnoses.*—Adenocarcinoma, salivary gland; cylindroma; adenoid cystic carcinoma; mucoepidermoid carcinoma; mixed tumor, basaloid; mixed tumor, malignant; adamantinoma; craniopharyngioma; adenomyoepithelioma.

Dr. Foote: Since the biopsy was reported, the patient returned here and a resection of the palate, axilla, antrum, and inferior orbital plate was performed. The tumor involved the soft tissue of the cheek overlying the wall of the antrum and invaded the inferior orbital plate posteriorly. It extended down as a large mass which had broken through the palate and presented itself within the mouth. There was a large polypoid tumor mass in the antrum. Combined with these operative findings is the past history of long continuous pain in the left infraorbital region. It seems a fair conclusion that the antrum represented the primary site of origin and from that location extensive local spread took place. Since the resection on January 5, 1952, there has been no clinical evidence of recurrence.

The majority made a diagnosis of some sort of malignant tumor. Whether benign or malignant, of course, is the crux of the issue. On the basis of the history alone, its malignant nature should be apparent. I rather expected that the runner-up diagnosis would be mixed tumor. Those who diagnosed mixed tumor variously qualified it by such terms as basaloma or basaloid, and one participant made a diagnosis of malignant mixed tumor. When you look at certain limited areas of the tumor, you can understand why these diagnoses of mixed tumor were forthcoming. Yet, there are give-away structural features of a specific group of tumors known to be malignant. They are characterized by a very long-term clinical course, culminating after years with the development of not only regional but generalized metastases.

The terminology employed for the diagnosis of this tumor was varied. The one most popular was cylindroma. That is reflected in current literature dealing with this group. I personally do not like it, one reason being that cylindroma has an air of mystery about it, and if you ask those who commonly use this term what it means, you get no standard answer. The majority, as far as I can judge, employ the term cylindroma to indicate an epithelial growth of cells around a central core. This is not the basic meaning of the word. The term cylindroma is an ancient one, employed by Billroth in an 1859 publication wherein he described a tumor of the antrum. Billroth was a young man of about 32 when he wrote that paper, and in using the term cylindroma he described cylindroid hyaline columns of acellular tissue which intertwined and were bordered by epithelial or connective tissue cells. Billroth was not referring to the epithelial components of his tumor when he used the term cylindroma. Some people use the term meaning a cylinder with nothing in it, some with mucus in it, and some with collagen in it, so the term is difficult for me to use.

Only one other pathologist besides myself decided to call this an adenoid cystic carcinoma, and this suggests that I should give up and start calling these things cylindroma too, but I do not think I shall. Adenoid cystic carcinoma was a term that Dr. James

Ewing used for many, many years, and having been under his disciplinary guidance, I have more or less gotten into the habit. I cannot say whether or not Dr. Ewing was the originator of the term.

It makes no particular difference how we report this type of salivary tumor as long as we do not report it as mixed tumor or something else benign. Reporting a thing like this as mixed tumor is dangerous. These are cancers. They are not locally recurring benign tumors. Look up the record of this type of salivary gland tumor. It is bad!

I do not know of a fully informing statistical account of the behavior of adenoid cystic carcinoma in the paranasal sinuses. One would hardly expect these lesions to be less malignant than those of the major salivary glands in which location we have good information on what to expect. The two largest series known to me are from the Mayo Clinic and from the Memorial Hospital. The Mayo series contains about 20-odd cases and the Memorial series a few more. The results of treatment in both series are pretty much the same. At the end of five years of follow-up in the Mayo and the Memorial material only 25 per cent of patients are clinically free of disease. That is not reassuring. The disease may cover a span of many, many years. There is one extraordinary case of a woman in whom the condition was diagnosed at age 15, treated at age 29, who still had a recurrence at age 80. Total duration of life of ten or more years from the onset of symptoms to death from disease is by no means uncommon. The tumors are not generally radiosensitive, but there are some noteworthy exceptions. In cases so far reported relatively conservative surgical measures have been the chief reliance in treatment. I believe the future will bring a better record of cure in these cases. More radical measures will be utilized and they can be fully justified.

(Lantern slides.) The adenoid cystic carcinoma, or cylindroma if you wish, has a great tendency to undergo hyalinization. On the way to that change intermediate stages are seen and are reproduced in these lantern slides. Incidentally, we have a good deal of difficulty in diagnosis of some frozen section specimens of adenocystic carcinoma. They can be hard to tell from mixed tumors. I do not believe we have made our last error as yet.

Dr. John J. Andujar of Fort Worth states that he has had some difficulty in using the term adenoid cystic carcinoma because surgeons are apt to think he is referring to an adenoid cystic basal cell epithelioma. I have only had one bad experience of that nature. That was some years ago, and it was in the reverse direction. I made a diagnosis of an adenoid cystic basal cell epithelioma in the nipple of a Negro man's breast; the surgeon thought I meant adenoid cystic carcinoma, and he did a radical mastectomy. I think that the objections your surgeons raise are logical, but after they raise the objection and you set them straight on it, I don't see why they give you any more trouble.

Dr. George Bailey, Memphis, Tenn.: Do these tumors metastasize widely?

Dr. Foote: They metastasize very widely, but that is apt to occur late in the course of the disease, years after it has been established, and usually is punctuated by multiple failures at local control. Of the people who die with distant metastases, 90 per cent have residual uncontrolled primary and recurrent disease.

Audience Member: From what tissue do you think the tumors arise?

Dr. Foote: I have never seen any clear study that gave the answer as to whether they were of ductal or acinar origin. I rather suspect that they are ductal tumors.

Audience Member: Would you comment more on radiotherapy of these tumors?

Dr. Foote: I would think of them as more radioresistant than radiosensitive, but on the other hand, they are at least moderately and sometimes impressively radioregressive. Remember, radioregressive does not necessarily mean radiosensitive. A radiosensitive tumor to me is one that regresses at a low dosage level. Many tumors will respond well after long continued treatment which naturally will go up to high levels of total irradiation.

Audience Member: What general classification of salivary gland tumors do you recommend?

Dr. Foote: In general I would not recommend any. The lot of the classifier, you know, is really difficult. When you classify, your soul lies naked and exposed. I start off with benign mixed tumors and an important subhead called malignant mixed tumors; then there is a group that I call mucoepidermoid tumors, and then the adenoid cystic carcinomas that are to most people cylindroma. After the adenoid cystic group comes a group of assorted adenocarcinomas that are varied in structural makeup, that go all the way from extremely anaplastic tumors through orderly or moderately atypical to highly malignant mucoid adenocarcinomas. The reason I put those together in a group of miscellaneous adenocarcinomas is that they are of such low frequency that one can scarcely get together enough cases to picture the natural history and response to treatment. The next group that I split off is termed acinic cell adenocarcinoma. I have been able to record about 25 of those, enough for a reasonable analysis. Then

comes the papillary cystadenoma lymphomatosum, the thing that I like to call a Warthin's tumor. I also use the term oxyphil cell adenoma for the tumor that is sometimes called oncocytoma. I separate oncocytoma and Warthin's tumor for various reasons. Then is included a list of lesions that have been variously reported under about seven different terms, recently published by Godwin under the term benign lymphoepithelial lesions. I do not include tumors of mesodermal origin because they are not indigenous to the salivary glands. They can occur in any location.

## CASE 2

*Diagnosis.*—Low-grade mucoepidermoid carcinoma.

*Contributor.*—Col. C. J. Lind, Brooke Army Hospital, Fort Sam Houston.

*Specimen.*—Parotid.

*History.*—A 25 year old Negro man noted a lump behind his left ear. The mass was excised, at which time a rather cystic central area was noted within one fragment.

*Submitted Diagnoses.*—Adenocarcinoma; mucoepidermoid carcinoma; mucinous adenocarcinoma; mucoepidermoid tumor (adenoma); mixed tumor; mucinous adenoma; mucocele or ranula.

Dr. Foote: Well, the majority have picked some sort of mucoepidermoid tumor. There is a split between those who called it malignant and those who called it benign. Therein lies the chief reason for presenting a tumor of this sort. I notice with some interest that a number of diagnosticians want to call this a mucous adenoma. That is understandable, because in the sections available there is practically nothing but a mucous cell component and in the section that I had myself it was extremely difficult to find anything that could be forced into an epidermoid structure.

When I see this type of mucous cell pattern, secretion, and arrangement, I just close my eyes and call it a low-grade mucoepidermoid carcinoma, whether or not the epidermoid element is good enough to pass on to my neighbor. If this tumor is seen later, at a recurrent stage, it might be found to be very epidermoid. So I think it is excusable to use the term mucoepidermoid when the epidermoid element is extremely attenuated. I also can understand the other point of view that a pathologist might not want to diagnose something he does not see. I cannot recall from 98 tumors of this family group, 1 with the structure of the current example which has yielded a node or distant metastasis; but there does occur the rare fatality due to inoperable local recurrence in the neck.

I would report this myself as a very low-grade mucoepidermoid carcinoma and add the comment that I have not as yet seen metastasis at this structural level and that the rate of recurrence in the group is roughly 15 per cent to date. That is subject to change. Mucoepidermoid lesions fooled us in 1945, and they may fool us again. We may see metastases at this level if enough time is permitted. I feel that the rate will always be very low, if it occurs at all. I would be opposed to calling this a mixed tumor because I would like to separate into specific types as many salivary gland tumors as possible. There ordinarily is no overlapping between mucoepidermoid tumors and mixed tumors. I know of one exception, a malignant mixed tumor, a tumor that yielded metastases which had an unusually bizarre structure. In one of the sections of the tumor that had areas of conventional looking mixed tumor was a little spot that was typical of the low-grade mucoepidermoid group. That is the only dilution that I have seen in mixed tumor by the mucoepidermoid. I would not want to report a thing like this as a mucocele or a ranula because that is too reassuring. This lesion has potential danger.

I would like to broaden the structural features of this group, which some of you may not have had the opportunity to see in full.

(Lantern slides.) Briefly, the gross specimen is highly characteristic of the lower grade tumors, most of which have a semblance of encapsulation. We try to separate mucoepidermoid tumors into three categories of ascending malignancy. In category 1 we have a five year follow-up record as follows: 16 cases, 13 cures, 2 recurrences, and 3 persons lost track of. In the second or intermediate group: 17 cases; 3 patients dead, 2 from metastases and 1 from local growth. In the third or highly malignant group the five year cure rate is about 20 per cent. It was of considerable interest to find that in the high-grade mucoepidermoid tumors the development of subcutaneous metastases was common, approximately 20 per cent of all cases. This usually prophesied an early fatal termination.

Dr. Leo Lowbeer, Tulsa, Okla.: Since you find no epidermoid elements in this tumor, why don't you just call it a low-grade mucin-producing adenocarcinoma?

Dr. Foote: I previously stated that it would be understandable why a pathologist would make such a diagnosis. I would not do it because experience has taught me that when you get a recurrence of this tumor, it is likely to be considerably epidermoid, and this tumor looks like it belongs in the family of the mucoepidermoid tumors.

Audience Member: What criteria do you utilize in deciding that a tumor has epidermoid elements?

Dr. Foote: I start thinking of squamous pearls, full of keratohyaline granules and intercellular bridges, then I just scale that down in my own mind gradually

until those qualities are lost, and then I admit I do not know myself. If we passed out a handful of mixed tumors and were asked to report how many had any epidermoid metaplasia, we would have a report that would range, say, from 5 to 30 per cent. The individual factor is widely variable. We report about 20 to 25 per cent squamous metaplasia in mixed tumors.

Capt. Leonard Paris, Fort Sam Houston: What is the objection to classifying tumors of salivary gland origin simply as either benign or malignant?

Dr. Foote: I would be afraid of doing that. To do so would mean putting a class of tumor which is conceded by conventional measures to yield a 75 per cent cure rate into the same pot with tumors that will yield a 75 per cent death rate. Lumping is an invitation to avoidance of specific accents in treatments. You don't treat every cancer the same way. If you adopt the diagnosis of benign or malignant, then the patient gets limited treatment, or he gets the works. There are some intermediate channels for both diagnosis and treatment.

## CASE 3

*Diagnosis.*—Chronic thyroiditis.

*Contributor.*—Dr. D. A. Todd, Nix Hospital Laboratories, San Antonio.

*Specimen.*—Thyroid.

*History.*—This white woman, aged 41, first noted swelling in the area of the right thyroid lobe one year ago, without evidence of toxicity. At operation a partially encapsulated specimen 6 by 4 by 3.5 cm. consisting of the right lobe and a portion of the isthmus was removed. The tissue was firm and reddish. The central area was largely occupied by a circumscribed but unencapsulated mass 3.5 by 2.5 cm. of similar color but more coarsely lobular than the remainder of the specimen. Three months postoperatively the patient showed signs of early myxedema.

*Submitted Diagnoses.*—Thyroiditis; struma lymphomatosa (Hashimoto); Hürthle cell tumor benign; Hürthle cell tumor malignant; hyperplasia; involutional thyroid; adenocarcinoma; Riedel's struma; adenoma benign; Gulf Coast goiter.

Dr. Foote: A few months ago Dr. Todd and Dr. Jackson were up at New York and eased into the laboratory with this case. I was taken with the structural features in the case and suggested that Dr. Todd submit enough material for the conference. I think I was having a bad day—one of those days when you feel like your mind would leave you if it hadn't gotten too weak to walk. I think we have made a tough selection. I was hoping when Dr. Todd got back that he would find evidence that the patient had a history of either long continued or interrupted episodes of hyperthyroidism, having seen this on a few occasions in thyroids with some of the structural features of the present case. I think at the time I stated that such a history was not always forthcoming, but that I even suspected that there was an obscured hyperthyroidism. Perhaps at that stage I had been reading some of the old publications of Alfred Scott Warthin, who had interesting ideas on the pathogenesis of the hyperthyroid state. But after we got further information there was a double backlash in the reel—not only did the patient not have a history of hyperthyroidism, but we found that this section came from a discrete nodule. I would not recall having this particular structural complex in any but a goiter that was mostly nodular or diffusely involved. As conference time came I could not make much out of this case. I was confident that the lesion was not a carcinoma, though I have seen such a diagnosis made on similar material. I sent a section to Dr. John B. Hazard at Cleveland and another section to Dr. William A. Meissner at Boston. I have statements from them that I will read in due course.

(Lantern slides.) The central panel is a topographical low power view. On the left are proliferative-looking thyroid cells in small groups and acini with an intermingling of principally lymphoid infiltrate but with a fair sprinkling of plasma cells. Looking at the diagnoses submitted makes one suspect that Al Todd just wanted to see a classification of thyroid disease. The submitted diagnoses have everything but cretinism and Himalayan goiter. I was relieved to see this because it is about the same way I had felt about it. So, we can go on now with opinions from different areas.

This is from Dr. Hazard:

I regard the lesion in this section of thyroid as struma lymphomatosa. I note that the lesion grossly was a circumscribed mass. There are several of these in our series. As far as I can determine, their course is no different from the recognizably diffuse or nodular type. On rare occasions the lesion is so definitely a localized enlargement as to be mistaken clinically for solitary adenoma; however, the follicular epithelium microscopically has shown a more generally oxyphilic change despite the gross appearance. We have not been impressed with the incidence of true hyperthyroidism, although I know some observers have described it. We have been impressed, however, with the incidence of anxiety state and hyperirritability in these patients.

Then we go to Dr. Meissner in Boston:

I looked over the slide representing the right lobe of the thyroid. I think we would diagnose a thyroid like this appearing in our routine material as a nonspecific chronic thyroiditis. There are some who would probably call it Hashimoto's disease, but I do not think it has a well enough developed picture to warrant this diagnosis. Glands having small follicles, acidophilic cells, large nuclei and a lymphocytic infiltrate such as this have also puzzled us as well as you, and we've always felt somewhat at a loss in explaining pathogenesis. I believe, however, that this represents an inactive or hypoactive thyroid state that may follow a previous hyperthyroidism, or which may develop without previous clinical thyroid disease. The fact that glands of this type are sometimes seen after a bout of hyperthy-

roidism has led some to believe that Hashimoto is the end result of hyperthyroidism. I have felt that this is not correct.

Dr. Meissner's comment is greatly interesting to me, and I was rather relieved to see that he thought this was at least a bit out of the ordinary. Dr. Hazard seems to have few cases of this sort and his cases follow the course of Hashimoto's struma. Dr. Meissner states that these cases are some kind of chronic thyroiditis. I do not know exactly how to resolve problems of this sort. The most important requirement here is to eliminate the diagnosis of adenocarcinoma. I must say that I am impressed with the fact that this patient did develop myxedema, and that would certainly be in line with the opinion Dr. Hazard expressed. Bear in mind that this patient did not have a total thyroidectomy, and it might mean that there was a strumatous lymphoid process in the residual lobe of this thyroid.

Col. C. J. Farinacci, Fort Sam Houston: Are oncocytes degenerative follicle cells, or do you believe that they are neoplastic?

Dr. Foote: In the first place, I am not entirely sure what an oncocyte is. An oncocyte is usually considered just a big, pink cell. I think it is an interesting speculation that these cells may represent worn-out members of functioning cellular groups. There is no direct proof of this. In the thyroid these large pink oncocytic cells are commonly called Hürthle cells. Personally, I'm an Askanazy man. If we went back to proper eponymic sources, we should eliminate Hürthle's name from use with the human thyroid. He did describe the cells that are named for him in the thyroid, but he found his cells in the interstices of the interfollicular portions of the canine thyroid, actually in newborn puppies. These cells disappeared from the puppy thyroid shortly after birth. It happens that Askanazy was a pupil of Hürthle, and in studying the human thyroid he found cells that Hürthle had described, and, no doubt, being a man of large soul and loyalty he named these thyroid cells in the human after the dog cells of his old master. Though it is a story of beautiful human relationship, calling these things Hürthle cells is not incumbent upon us.

Dr. William W. Tribby, Memphis, Tenn.: Isn't struma lymphomatosa a form of chronic thyroiditis?

Dr. Foote: It is so regarded by several groups, disregarded by other groups. I think the Cleveland group votes thyroiditis on it; in the Meissner-Warren fascicle they discuss three forms of thyroiditis—struma lymphomatosa, Riedel's struma, and subacute thyroiditis. Here you go into a difficult area of defining what actually constitutes an inflammatory process and how you separate it from a noninflammatory infiltrative process.

Dr. Leo Lowbeer, Tulsa, Okla.: Dr. C. A. Hellwig from the Hertzler Research Foundation in Halstead, Kan., believes that in Hashimoto's disease there is an abnormal colloid produced because of an abnormality of the thyrotoxic hormone of the pituitary gland. He has found significant differences between this abnormal and normal colloid under the electron microscope. He thinks that the abnormal colloid is taken up by macrophages, often found in follicular lumens, and that it stimulates marked lymphocytic reaction. According to that concept, there is, then, an inflammation due to an abnormal endocrine product.

Dr. Foote: In other words, a secondary inflammatory process on a basic metabolic basis. That is an interesting comment to make in this case. In Dr. Meissner's statement, which I did not read in full, he wondered if these lesions were not on a hormonal basis, perhaps pituitary imbalance, which we are not as yet able to recognize.

## CASE 4

*Diagnosis.*—Papillary adenocarcinoma of thyroid.

*Contributor.*—Lt. Col. F. M. Townsend, USAF Hospital, Lackland Air Force Base.

*Specimen.*—Thyroid.

*History.*—This white girl, aged 17, gave a five months' history of enlargement of the right thyroid lobe without evidence of toxicity. At operation the lobe weighed 35 Gm. and measured 7 by 3 by 2 cm. It was covered by a shaggy fibrous capsule. On section the tissue was firm, yellow-tan, and separated into indistinct lobules by fibrous septums.

*Submitted Diagnoses.*—Carcinoma; papillary adenocarcinoma; papillary adenocarcinoma and thyroiditis; papillary adenocarcinoma and Hashimoto; small cell carcinoma; struma lymphomatosa; chronic thyroiditis; Riedel's struma; nodular goiter.

Dr. Foote: We have another thyroid situation. The microscopic pattern is mainly a papillary and proliferative looking process with abundant calcific concretions. We find in the sections a considerable lymphocytic and other round cell infiltrate. The vast majority favor carcinoma. That is my diagnosis—papillary carcinoma of the thyroid. Struma lymphomatosa has been given second most important notice. I believe we can exclude that diagnosis here since proliferatoin is not characteristic of that disease. I do not recall seeing or having read a report of the formation of multiple psammomatous bodies in cases of struma lymphomatosa. I also would eliminate chronic thyroiditis and Riedel's struma for similar reasons. We must recognize the presence of carcinoma in this thyroid. That, for the most part, was accomplished.

Now people will ask, "How do you call that carcinoma?" saying the appearance is innocent and uniform, and then they will say there are not many

mitoses, and so forth. Well, the way I would answer that is simply to state that the disease must be studied on the basis of its behavior as well as on its microscopic aspect. There are many lesions that look one way and behave another. Thyroid lesions of this sort, for the most part, are sooner or later associated with regional or distant metastases, and hence justify the diagnosis of papillary carcinoma.

We have 8 diagnoses combining papillary carcinoma with Hashimoto's disease and thyroiditis. That is a fully justified dual diagnosis to make. It has a precedent already, and we can cite the recent article by Crile and Fisher in *Cancer*. They described 2 cases of papillary carcinoma, and in 1 of these they thought there was also subacute thyroiditis, and in the second, struma lymphomatosa. In 1 of the 2 cases they felt positive, and in the second they were not sure, that the lymphoid tissue was not reactive to the presence of tumor. But in any event the pathologists who selected the dual diagnosis have the support of that paper and perhaps they have similar cases in their own material. I cannot recall in the rather abundant Memorial Hospital material having seen as extreme a degree of lymphoplasmacytic infiltrate as we have in this case of Colonel Townsend. Dr. Klinck at the Armed Forces Institute of Pathology some years ago made a careful study of the frequency of the lymphoid component in papillary thyroid tumors, but I do not recall from his report so extreme a degree of this change. I judge that this is another diagnostic area where time must work its slow way before standardization can be achieved.

Ordinarily one thinks of Hashimoto's struma as essentially uncomplicated by other thyroid disease. Joll emphasized that he did not have in his large personal series of cases of struma lymphomatosa any significant crossing with other thyroid lesions. He even went so far as to say that he scarcely had any combined with nodular goiter. We at Memorial Hospital could not go quite that far in our material, but we almost could. But why not accept readily the dual diagnosis here?

In regard to the basic diagnosis of papillary carcinoma, I would like to expand on the structural features of this disease just a bit. Papillary thyroid carcinoma, generally speaking, is rather characteristic. In the past its relatively innocent looking structure has been responsible for the common usage of the term papillary adenoma, but this seems to be on the decline as does papillary cystadenoma. For example, in the Boston material, not many years ago, papillary adenoma was a frequent diagnosis; in the most up-to-date summary of that material I noticed that only 4 and a fraction per cent of all the adenomatoid lesions of their thyroids is classified as papillary adenoma. In the more recent material from the goiter registry there is a perceptible decline in the usage of that term, so I think it is largely disappearing. It took a long time to establish that certain relatively innocent looking papillary lesions in the thyroid were capable of malignant clinical behavior.

Going back to the structural features of these papillary thyroid carcinomas, we hear altogether too much comment on just their papillary components. Actually, it is difficult to find a thyroid carcinoma that is purely papillary. Almost without exception there are other structural features, and the follicular component as seen in the extreme left lantern slide is common. This follicular component is frequent in lymph node and distant metastases of the tumors.

Then there are solidly growing areas in papillary carcinomas. So far as I can judge from studying the clinical records of our cases, the appearance in the papillary group of the follicular and more solidly growing areas has no bearing on the clinical course or the response to treatment. Our old case records of the 'thirties and 'twenties show that structures like those in the slide were commonly put down as grade 2 and sometimes as grade 3 thyroid carcinoma. We know now that that was an error and presently call the entire group carcinoma, grade 1, because of the renowned sluggishness and slow clinical maturation of the disease.

Primary papillary carcinomas and their metastases may vary a lot in structural dominants. One may have in the thyroid a tumor that is predominantly papillary and study the lymph node metastases to find that follicular areas are more abundant. One might find the reverse.

Dr. John B. Frerichs, San Francisco: I wonder if the remainder of this thyroid was studied?

Dr. Foote: I wanted to know that, too. What you would like to know is that a piece of thyroid uninvolved by cancer did show typical Hashimoto's struma, isn't it? Exactly. That seems to me to be an ultimate requirement before the diagnosis of carcinoma in Hashimoto's struma is totally accepted. The point that you make is very apt.

Dr. A. O. Severance, San Antonio: What are your thoughts on irradiation therapy in cancer of the thyroid?

Dr. Foote: I am not qualified to give a detailed discourse on that, but a few generalities can be safely expressed. I would not propose irradiation therapy as a means of primary cure of thyroid cancer. The one form of thyroid cancer that uniformly gives good radioregression is the group of papillary tumors. Inasmuch as the papillary tumors constitute approximately 50 per cent of the total, that is indeed for-

tunate. There are in the Memorial Hospital records a small number of long term cures of papillary carcinoma in which the result was effected solely through irradiation. These are exceptional. A great many cases whether operable or not will yield good regression.

Dr. E. L. Frazell of New York, who knows this aspect much more intimately than I, tells me that it is rather unpredictable just which case is going to give a maximum response. He further states that over the long pull the frequency of local recurrence is greater than he wants to cope with. The Head and Neck Surgical Service at Memorial Hospital employs radical surgery in the effort to control even the papillary carcinomas of the thyroid. Radiotherapy is used as a palliative means and with useful results in a good many cases.

## CASE 5

*Diagnosis.*—Metastatic islet cell tumor from pancreas.

*Contributor.*—Dr. Donald Penner and Dr. T. H. Williams, Winnipeg, Manitoba, Canada.

*Specimen.*—Mass from mediastinum.

*History.*—This white man, aged 28, was hospitalized with a severe chest cold, cough, headache, chills, and pain in the right lower thorax. Roentgen study revealed a circumscribed, spherical mass in the right anterior thorax extending into the right lung field from the mediastinum. At surgery a 14 cm. mass was found extending out from the right side of the mediastinum at the level of the aortic arch. A second mass, 4 cm. in diameter, was present just above the first. The larger mass was encapsulated, and its cut surface was variegated, predominantly white, with numerous areas of hemorrhage and necrosis.

*Submitted Diagnoses.*—Metastatic carcinoma; metastatic carcinoma from lung; metastatic carcinoma from thyroid; metastatic carcinoma from liver; mesothelioma; aortic body tumor; neuroepithelioma; sympathicoblastoma; teratoma; malignant thymoma.

Dr. Foote: We come to case 5 from Canada, surgical material furnished by Dr. Penner and, we have a surprise for you, the autopsy material from Dr. Williams.

I got the impression from the diagnoses that this was a difficult case. Let me tell you how this case came into my life. I want to say in advance that I think in this audience there are at least two geniuses: those who diagnosed pancreatic carcinoma. In 1945, when Dr. Fred Stewart was on one of his vacations, the sections from this case were sent by Dr. Penner. Dr. Penner thought the sections suggested a bronchial adenoma, and he saw an area which reminded him of salivary gland tumor. I wrote to Dr. Penner agreeing that there were features of so-called bronchial adenoma, somewhat more aggressive looking, however, than usual, and then made a suggestion that we could have a malignant pleural mesothelioma, admittedly largely on the basis of position. And then —these are old notes from correspondence—"I doubt if this patient remains well." When Dr. Stewart came back to the laboratory, he looked at the sections and said he was inclined to a diagnosis of paraganglioma. That accent, I see, is reflected in some of the members of this body because we have diagnoses of aortic body tumor and the mysterious chemodectoma.

Matters went along without further disturbance, until 1948. The patient developed further symptoms for which reexploration was done, and at that time another mass of tissue was removed from essentially the same location as the first. We were sent sections from it and we estimated them as follows:

> As you know, we were puzzled . . . in 1945, and we could really make no diagnosis, but would resort to desperate suggestion. The recently received slide is essentially identical with those first seen. At the present time we don't think we can give any real support to the two diagnoses formerly made. We would like to suggest, however, two possibilities: one, that the tumor is metastatic from an islet cell tumor of the pancreas, or two, that it is metastatic from a carcinoid of the gastrointestinal tract. We can undersand how you will recoil violently from these suggestions. Recently, however, we had a section of a cervical node from a patient who for eight years had a very bulky mediastinal mass. Structurally, this section has a good deal in common with the submitted case.
>
> In the recent case we made three suggestions: metastasizing bronchial adenoma, islet cell tumor, and metastasizing carcinoid. At autopsy we found a 7 cm. tumor in the tail of the pancreas, pressing on the stomach and filling up the liver with metastases. This could only be a primary islet cell tumor. The long duration of the mediastinal mass was considered consistent with the known behavior of some of these islet cell tumors. We don't think that we'll ever get an answer in your case until an autopsy is done.

In July, 1952, we had a letter from Dr. Williams which I will summarize. He said that he had recently performed an autopsy on the patient, and the final diagnosis was metastatic nonfunctioning islet cell tumor of the pancreas. He found the pancreas almost universally involved with tumor, and there had been an extraordinarily wide dissemination of the lesion in the lymph nodes, viscera, and lungs; scarcely an organ was uninvolved.

So, in this case we have to pass the palm to the two pathologists who picked this as a metastatic pancreatic tumor, an extremely penetrating diagnosis.

The sections were sent to Dr. Virginia Frantz of New York, who has outstanding experience with islet cell tumors. I asked her to comment on her records of cases of long term survival in which metastases had been demonstrated prior to autopsy. She stated that the long survivals were in patients with nonfunctioning tumors. She cited 1 patient who lived for five years after a positive biopsy of liver metastases and a second who lived for four and one-half years after liver involvement. She also referred to

cases described by Dr. Howard in his extensive 1950 *Surgery, Gynecology, and Obstetrics* article in which there were 2 patients, 1 who lived for four years and 1 six years after metastases were demonstrated. Hence, the extremely sluggish clinical behavior of the case under consideration is reflected in other reports.

## CASE 6

*Diagnosis.*—Terminal bronchiolar or alveolar cell carcinoma.

*Contributor.*—Dr. J. M. Moore, Santa Rosa Hospital, San Antonio.

*Specimen.*—Lung.

*History.*—This white man, aged 48, was admitted to the hospital with symptoms of shortness of breath and inability to void. There was a previous history of pneumonia with hemoptysis, cough, and "wheezing" which persisted after antibiotic therapy. Roentgenograms showed diffuse infiltration of both lungs. Pleural effusion which became bloody developed. At autopsy the right lung weighed 700 Gm., the left lung 1,300 Gm. Cut section through both lungs exhibited small, firm, gray nodules varying from 0.1 to 2.5 cm. The remainder of the lungs on cut section had a mottled red-gray appearance.

*Submitted Diagnoses.*—Alveolar cell carcinoma; bronchiolar carcinoma; pulmonary adenomatosis; papillary adenocarcinoma, metastatic; bronchogenic adenocarcinoma.

Dr. Foote: Only two suggested the possibility of a metastatic tumor. I think that is a rather prudent proposal since a few kidney cancers may yield lung metastases that cannot be distinguished from the present section.

I think we can quickly eliminate several diagnoses proposed, because we have one structural feature that must be given attention, namely, these psammoma bodies. With psammoma bodies on the scene one would think immediately of a primary lesion in the ovary, but the patient's gender relieves us of this set problem. Even if the patient were female, I would be inclined to eliminate it because we expect psammomatous carcinomas of the ovary to fill up the lungs. I have never seen an exception to that.

Another psammoma-forming tumor would be papillary thyroid carcinoma. These may involve lungs extensively without showing much evidence of disease elsewhere, but we have sound grounds here for eliminating thyroid carcinoma since one can find plenty of cells in this section that are mucus-containing. Papillary thyroid carcinomas do not contain mucous cells. I have had that question come up from time to time in diagnostic debates and have stained a good many papillary thyroid tumors unsuccessfully for mucus. To gather reinforcement for that opinion I wrote Dr. Allen Graham of Pittsburgh a few years ago and asked him his experience. He said he had never been able to demonstrate mucus in his papillary tumors. A few mammary carcinomas have psammoma bodies and sometimes there are a few in the prostate.

There is one other site, though, that can produce metastatic papillary tumors with psammoma bodies and hide away a primary lesion. That is the kidney. We had 2 kidney tumors that first came to attention through the medium of lung metastases. In the first case the primary tumor was found at autopsy. In the second we had made a diagnosis of terminal bronchiolar carcinoma. There was a history, however, of nephrectomy eight years before death, and when these old slides were obtained, there was the primary tumor —papillary and psammomatous.

The diagnoses of pulmonary adenomatosis should be eliminated. Cytologically these tumor cells are malignant, and tumors like this are known to yield metastases. Pulmonary adenomatosis per se should be a nonmetastasizing lesion. I am well aware that in the literature rare examples of metastasis are reported in pulmonary adenomatosis, but I suspect confusion in some.

A principal reason for including this case was to present some findings from the Memorial Hospital material on this subject that are at variance with the general concept of origin of alveolar cell or terminal bronchiolar tumors. Almost without exception (and I know of no article that pointedly denies it) these lesions are considered to be of multicentric origin. I was much impressed from a recent study of 22 cases that much of the apparent multicentricity of these lesions is explainable on the basis of peribronchial, perivascular lymphatic, and subpleural lymphatic spread. The vast majority of material reported to date on this lesion has been autopsy material which represents necessarily the terminal phase of the disease. Most of our surgical specimens are different from the ordinary concept of multicentric alveolar cell tumors. Notice in the central lantern slide a pneumonectomy specimen; this is an extensive tumor infiltrating here and there, sort of circumscribed and yet nodular at its edges, with satellite nodules. There are scattered metastatic nodules and not independent primary foci. I have seen at autopsy lungs with the type of lesion distribution that was shown in Dr. Moore's present case, but out of the original 22 tumors that I examined, 7 were uninodular. At any rate, not all of these tumors are multicentric and would not appear so if they were seen earlier in their course. At least some of them are capable of successful control by surgical measures.

Without adequate grounds some authors will state that these tumors represent but malignant variants of pulmonary adenomatosis. They are certainly not absolutely dependent on preexisting pulmonary adenomatosis because we have case after case in which there is not the faintest question of the coexistence

of pulmonary adenomatosis. If this type of bronchiolar carcinoma or alveolar cell tumor is one that grows out of preexisting pulmonary adenomatosis, why aren't there more cases of pulmonary adenomatosis? Where a cancer traces back to a benign lesion, the frequency of the benign lesion overshadows that of the malignant. What is the situation with pulmonary adenomatosis and alveolar cell tumor? As I recall, in 1941, Neuberger accepted 25 cases of alveolar cell carcinoma. Human pulmonary adenomatosis is much less frequently reported. Over a twenty year period at Memorial Hospital we had 32 cases of alveolar cell carcinoma and 1 case of pulmonary adenomatosis. I think the relationship of the two lesions is greatly exaggerated.

## CASE 7

*Diagnosis.*—Pigmented ganglioneuroblastoma.

*Contributor.*—Dr. Ellis Kellert, Schenectady, N. Y.

*Specimen.*—Mass from mediastinum.

*History.*—A routine chest roentgenogram of an asymptomatic 52 year old man exhibited a mass in the mediastinum. At operation a mass 12 by 9 by 8 cm., weighing 211 Gm., was removed from the upper left side of the chest in the angle between the ribs and the spinal column. It was partially encapsulated, and segments of ribs and skeletal muscle were attached. The cut surface was soft, yellowish, mottled, and streaked with white tissue.

*Submitted Diagnoses.*—Ganglioneuroma; mesothelioma malignant; sarcoma (leiomyosarcoma, liposarcoma, xantholiposarcoma, fibroliposarcoma); teratoma, teratocarcinoma; carcinoma, metastatic from kidney; xanthofibroma; xanthogranuloma; melanoma, malignant; myoblastoma, malignant; chromaffinoma, paraganglioma; meningioma, malignant; chordoma; hibernoma; xanthomatous giant cell tumor of bone.

Dr. Foote: The various cell types in this tumor require comment. The first to be mentioned are the plentiful, fat-filled phagocytic cells, which I think it reasonable to assume are secondary scavengers. The next cell type includes the larger cells, those usually with a single nucleus but sometimes with two or more. The third type, not particularly well shown at this magnification, is similar to and not much larger than lymphocytes. The larger cells, when examined closely, show clearly the presence of brown pigment, and this calls to mind a case reported by Millar in the *Journal of Pathology and Bacteriology* in 1932. He recorded a tumor that was composed of large ganglion and small neurocytic cells. Some of the ganglion cells contained melanin, and hence he had a unique melanotic ganglionic tumor. His lesion was malignant. It metastasized. It lay in the thorax much in the same position as in the current case. Too quickly we reported this tumor as probably a counterpart of the Millar tumor, but made the proviso that this would depend upon demonstrating that the brownish pigment was melanin.

Look at that pigment closely. It really does not look too much like melanin, and it isn't. By resorting to special stains we found that that pigment is probably lipochrome, positive with scarlet red, and failing to blacken with silver treatment. Going a bit further, we tried to develop evidence of the neurogenic nature of the tumor. We did several silver impregnations and were satisfied with what we saw in the Bodian impregnated material. The tissue that we had was formalin fixed, and we were sorry that we had no chloralhydrate material since that is a great aid in carrying out certain impregnations, particularly the Cajal method. Some of these large cells have silver impregnated processes coming off in a tennis racket–like fashion. A good many of the small cells in the silver preparations had tails of one sort suggesting neurofibrils.

Several of us in the laboratory were trying to force an axone accent into these structures, but felt unsure about it; so, knowing his large experience in matters of this sort, I got Dr. Nathan Chandler Foot of New York to examine the sections. He said he doubted that he could demonstrate true axones; that the long processes of the large cells he liked to refer to as taproot structures; and that they typified ganglionic cells. His comment suited us very well since it supported what we were trying to establish, that this was an odd variant of the neurogenic series. We decided to interpret the smaller cell elements as relatively undifferentiated neurocytic cells and the larger cells as poorly formed ganglion cells; hence, we wound up with a diagnosis of pigmented ganglioneuroblastoma.

As regards the submitted diagnoses, I do not know how one could settle the issue as to whether or not this is a malignant tumor. I feel certain that it is of nervous origin, and the latter seems to have been the dominant thought of this group. Mesothelioma was suggested, but I believe that the pigment and the ganglionated aspect of some of these cells would successfully rule that out. Various types of sarcoma were suggested. Teratocarcinoma—I could not find any carcinoma in this case. Metastatic from kidney and other metastatic carcinomas—if you are not satisfied with structural explanations, the autopsy showed no other source of tumor. Xanthofibroma and xanthogranuloma—these diagnoses recall the cases that Dr. Heuer reported some years back: xanthomas of the mediastinum which on retrospect seem likely to have represented extremely xanthomatous intrathoracic neurilemomas.

We looked carefully in the sections at hand, and we got multiple blocks from Dr. Kellert in the hope of turning up additional neurogenic evidence. We

were looking for something that looked like plexiform neurilemoma or a Schwannian type of structure. We could not find it. Hence we wound up calling this a pigmented ganglioneuroblastoma, feeling, of course, some uncertainty as to whether or not it was a truly malignant tumor.

I would like to ascertain if any of you have a different or corroborative slant on this tumor, skirting for the moment the unsettleable problem of whether or not it is malignant. We have shown these sections to a good many pathologists visiting through our area, and we have not found anyone who felt at home with this tumor.

## CASE 8

*Diagnosis.*—Eosinophilic granuloma.

*Contributor.*—Dr. L. J. Manhoff, Jr., Robert B. Green Hospital, San Antonio.

*Specimen.*—Stomach.

*History.*—This Latin-American woman, aged 62, complained of frequent vomiting without pain or hematemesis. Roentgen-ray revealed a prepyloric gastric polyp. A gastrectomy was performed.

*Submitted Diagnoses.*—None. Slides not sent to pathologists.

Dr. Foote: The gross specimen showed the polypoid character of the lesion, covered by mucosa and nonulcerated. The histologic section showed a granulomatous process punctuated with frequent eosinophils, and in the central panel those cellular elements are seen at higher power.

I wonder how many of this group have had a case of this sort? It is a rare bird. At a meeting of about 125 pathologists in New York there were only two who had had a case of this sort. We have had only a single example of this in our own routine material. I have seen about 6 or 8 cases from hither and yon. I have not seen a report of this type of lesion involving another part of the gastrointestinal tract, with the exception of one rumor that there was a case reported from the small intestine. That case I have been able to find. The largest single report seems to be a series of 6 that Dr. Vanek reported in the *American Journal of Pathology*, 1949, as I recall. Most of the reports deal with single cases, or maybe two.

The lesion is usually not large, characterized principally by submucosal growth with more or less involvement of the muscular coat. Ordinarily the mucosa is not ulcerated and now and then the lesion is polypoid, as in the present case. Two of the 6 lesions that Vanek described were polypoid, and with this setting we may get a radiologic diagnosis of polyp. I believe that was true in this case. Yes, prepyloric polyp was diagnosed. These lesions are not ordinarily incidental findings, but they are associated with some sort of gastric symptomatology. Some of them are so vague that it seems to me they could be put under the heading of epigastric bewilderment of varied sorts. Others seem to have a symptom complex suggesting ulcer. The lesions have uniformly been controlled by local surgical procedures in cases reported to date. The literature is weak in long-term follow-up cases, however.

There is no great difficulty in the differential diagnosis of this lesion. It looks a good deal like the lesions that we know as eosinophilic granuloma of bone or Hand-Schüller-Christian disease, but there is a near or total lack of the vesicular, umbilicated, or notched histiocytic cells found in those conditions. There is no association between this gastric lesion and similar lesions elsewhere in the body. In no reported cases are bone abnormalities noted. The majority of these patients do not give any history of allergic states. There are 2 reports that give exceptions to this, but to date allergic phenomena are not impressive.

## CASE 9

*Diagnosis.*—Malignant melanoma.

*Contributor.*—Dr. D. A. Todd, Nix Hospital Laboratories, San Antonio.

*Specimen.*—Gallbladder.

*History.*—This white woman, aged 28, was seen with epigastric and right upper quadrant pain when seven months pregnant. She complained of chronic nausea, loss of weight and appetite, some pain and numbness in the right arm, and episodes of acute illness lasting up to eight hours. At operation a thick-walled gallbladder, 9 by 4 cm., with a smooth serosa, was removed. A soft, friable, 7 by 2.7 cm. fungating tumor with areas of necrosis over the surface almost filled the lumen. Cut surface was solid, of a glistening gray-white color. Several small stones were present. One month later craniotomy confirmed symptoms of a frontal lobe tumor.

*Submitted Diagnoses.*—Malignant melanoma; carcinoma (mucoepidermoid, stomach, bronchogenic, gallbladder, choriocarcinoma); sarcoma (reticulum cell, leiomyosarcoma, liposarcoma, fibrosarcoma, Schwannian); medulloblastoma; embryonal hepatoma; angioendothelioma; malignant neoplasm.

Dr. Foote: This was a polypoid lesion, elevating but not ulcerating the mucosa and not showing any traceable continuity with or origin from epithelium. I was relieved to find the popularity of malignant melanoma. I had anticipated that we would get a good many diagnoses of leiomyosarcoma and of anaplastic carcinoma. There is not much else in the cancer line that can happen to gallbladder beside those things, except a tumor that came from somewhere else. I made the diagnosis of malignant melanoma in this case and thought it was metastatic. The rarity of each of the diagnoses submitted is considerable, even including carcinoma, which we do not expect in the gallbladder at the age of 27. Dr. Todd secured a history of prior removal of a cutaneous melanoma.

The frequency of primary malignant melanoma of the gallbladder is impossible to state, but it must be about zero. I have a friend in Albany, N. Y., who

thinks he has one. I think he probably does not have one. He showed me the sections about four years ago and I called it a malignant melanoma, but I cannot remember saying it was primary. I talked with this Albany surgeon about a week ago, but he could not get the slides down again before I took off. It is an interesting case anyway, and I will quote it briefly.

Dr. Tom Walsh did a cholecystectomy on an adult male in 1940 following an attack of gallstone colic. About four weeks before this attack of gallbladder colic the patient had had a nodule removed from deep within the jugular lymph node chain. The report on that specimen had not caught up with the patient by the time he began to get a stomach ache, so the pathologic diagnosis was unknown to the surgeon at the time of laparotomy. When that lymph node was reported, it was metastatic melanoma. In the gallbladder of the patient were 2 polypoid masses, and sections of each of these are nonpigmented melanoma. The man had absolutely no external or historical evidence of mole, cauterization, or fulguration, or anything, so they waited for things to get worse, but the patient prospered and is hale and hearty thirteen years afterward.

One may argue that here is a primary melanoma of the gallbladder with a cervical node metastasis with good reasons until he begins to strain the issue. In the first place, an acceptable primary malignant melanoma of the gallbladder is hardly to be found. Dr. Walsh tells me that he could locate only 4 allegations of primary melanoma of the gallbladder in a search of the literature for many years. Only 2 of those have been reported in the last twenty-five years.

I suspect that Dr. Walsh's case is of metastatic melanoma, and the reasons for it are these: To get finite evidence of primary origin of a melanoma ultimately rests upon the demonstration of suitable cells of origin, and we would like to see junction changes. Secondly, the lesions in this gallbladder were multiple. That is not unheard of in melanoma, of course, but is off the beaten path. In the third place, malignant melanoma has a taste for metastasizing to the bowel mucosa, almost anywhere along it, gallbladder very infrequently, to be sure, but nevertheless it can exist. Furthermore, almost every surgeon or internist has had in his practice this or that case of undoubted malignant melanoma in which no primary lesion was ever discoverable. It is also true that metastases of melanoma upon occasion apparently have traced back to seemingly quiescent moles. Not too long ago we had such a case in which a lesion scarcely more than a freckle turned out to be a perfectly acceptable melanoma histologically. Another fact to be considered is the rare spontaneous dissolution of primary malignant melanoma. There is a wonderfully well documented case reported in *Cancer* by Dr. Wilbur Sumner of Jacksonville, Fla.

Going a little bit farther with this melanoma business of the gut, the affinity of melanoma metastasizing to the gastrointestinal tract seems to have been largely overlooked. But I find without too much trouble that Dr. Peter Herbut and a co-author were rather impressed with it in 1945. They recorded 25 cases of melanoma involving the small intestine and concluded that all the tumors were metastatic. We found in our own autopsy material involvement of the gastrointestinal tract in about a third of the cases. I was talking over this case with Dr. Stewart before I came down. He said he would put melanoma in second position as the one tumor most likely to metastasize to the intestine after malignant lymphoma. There may be some objections to the absence of pigment in this lesion, but insisting on pigment in melanoma is just too much. There would never be a problem with melanoma if the lesions were not often nonpigmented.

## CASE 10

*Diagnosis.*—Leiomyosarcoma.

*Contributor.*—Col. C. J. Lind, Brooke Army Hospital, Fort Sam Houston.

*Specimen.*—Ileum.

*History.*—This white man, aged 27, was admitted to the hospital with right lower quadrant pain which was diagnosed as caused by a ruptured appendix. He was treated with antibiotics and discharged to return in six weeks for surgery. He returned in three weeks with continued discomfort. At operation the appendix was normal. A segment of ileum was removed; it contained a moderately firm, 4.5 cm. circumscribed oval mass. The external surface appeared encapsulated. On cut section the tissue varied in appearance from fatty to fibrous, with areas of softening. One area of the mass appeared to be firmly adherent to, or arising from, the muscularis.

*Submitted Diagnoses.*—Leiomyosarcoma; myosarcoma; leiomyoma; neurofibroma; fibrosarcoma; regional ileitis.

Dr. Foote: The history as distributed brings you down to the time of resection, and we can supplement these data with other developments. We find now that this patient, over a three year period, had had three attacks of what was characterized as severe gastritis. During this interval of several weeks when he was on antibiotics he gave a three weeks' history prior to operation of passing tarry stools for the first time. That sort of a history in a patient who has abdominal complaints of one sort or the other is a warning that a malignant connective tissue tumor of the stomach or intestine should be considered. We will have some illustrations of the mechanism of this in a few minutes. The sections distributed are representative of the findings in the surgical material. Major Sproat, will you give us a follow-up history?

Major Harry Sproat, Fort Sam Houston: The surgical specimen was removed in January, 1949; subsequent to this the patient followed a downhill course, and an autopsy was performed at this station in January, 1953. Autopsy showed the usual signs of terminal carcinomatosis. Examination of the organs revealed the intestinal tract, the liver, and the lung to be filled with tumor nodules. This was particularly true of the intestinal tract. Some of these were pedunculated, and those that were pedunculated exhibited necrotic centers.

Dr. Foote: Thank you, Major. The majority voted sarcoma, with a slight margin of two. We could anticipate the problem here was going to be whether or not the degree of anaplasia in this surgical specimen was sufficient for a diagnosis of leiomyosarcoma. I think that that would depend much on whether or not one has had enough personal trouble with such a lesion to realize that it is capable of running a malignant course. I have become skittish about smooth muscle lesions of stomach and intestine, including rectum, if they look the least bit different from the most innocent looking myoma.

We had years ago at the University of Virginia 2 rectal cases, and on the local lesions removed from the rectum we made diagnoses of leiomyoma. The lesion in the first of these cases recurred after about two years, and was inoperable when the patient came back. We got a biopsy of his recurrent tumor looking scarcely any different from the one that we had called a leiomyoma. The other rectal patient, after six months, got an orbital metastasis, and this looked just like the local lesion we had called a leiomyoma. This was in 1937 or 1938, and I sent the first lesions up to Dr. Fred Stewart at that time. He wrote back a typical long-winded letter of about two lines, saying he had learned to respect low-grade looking, smooth muscle tumors of the gut, period! So I had a rather early and abrupt indoctrination into the evil doings of these relatively well differentiated smooth muscle tumors. We do not hesitate to call lesions of this sort low-grade leiomyosarcomas.

Grossly, especially in the stomach, these leiomyosarcomas are ulcerating, projecting tumors. The ulcerations are sometimes multiple. Typically the ulceration is central, on a hillock, and deeply excavated, thus accounting for the repeatedly observed clinical phenomenon of sudden massive hemorrhage. In addition to the bulging ulcerated form, there is the pedunculated, polypoid form which may lie within the cavity of the stomach. There also may be the externally presenting type in which the lesion becomes polypoid and projects through the serosa. These external tumors can assume enormous proportions, 25 and 30 cm., and at that size they are, of course, commonly broken down and cystic.

Col. C. J. Lind, Fort Sam Houston: Dr. Foote, are you as malignant in diagnosis in uterine leiomyomas as you are in intestine?

Dr. Foote: I think not, but I have learned the spirit of caution in these uterine lesions that are generally referred to as cellular myomas. Goodness knows, nobody can standardize the histologic level at which a diagnostic take-off occurs. Nobody can standardize it for himself, let alone other people. Everyone has to go on the basis of what has happened under his own eyes. These things come into our consciousness by our making a mistake on them, and these are valuable experiences if not forgotten. I have seen 3 uterine tumors that were debatedly diagnosed here and there as cellular myoma versus leiomyosarcoma, and each of the 3 patients died as a result of disease. All of them died from dissemination of intraperitoneal disease or inoperable local recurrence with urinary obstruction. Each of the 3 women had had a supracervical hysterectomy. I rather suspect that if they had had complete hysterectomies they might have escaped the recurrent tumor. So I would be a bit more inclined to fear a smooth muscle tumor of the gut that showed limited dedifferentiation.

Dr. Leo Lowbeer, Tulsa, Okla.: We had occasion to observe 3 cases of leiomyosarcoma of the ileum which showed marked variations in behavior. In 1 case, that of a 50 year old man, a biopsy specimen was taken from a mesenteric mass which was diagnosed by me as a very cellular leiomyoma, probably leiomyosarcoma. Slides were seen by many prominent consultants, but only Ewing concurred in the diagnosis of low-grade leiomyosarcoma; the others including those from the Mayo Clinic considered it a leiomyoma. The patient did not consent to a complete removal of the tumor at that time. Five years later he returned with symptoms of an intraperitoneal abscess. On operation a large perforated tumor of the ileum was found and resected; it proved to be a low-grade leiomyosarcoma, perforated into the lumen as well as into the peritoneum. There were no metastases. The patient died five days after operation. No autopsy was obtained.

The second case was that of a 55 year old woman who gave a history of a transient episode of melena and of an abdominal lump, noticed only one month before. Such lump was palpable, and on exploratory laparotomy ascites was found, together with an enormous nodular liver and countless tumor nodules in omentum and peritoneum. These were subjected to biopsy and tentatively diagnosed as a leiomyosarcoma of moderate morphologic malignancy. The patient

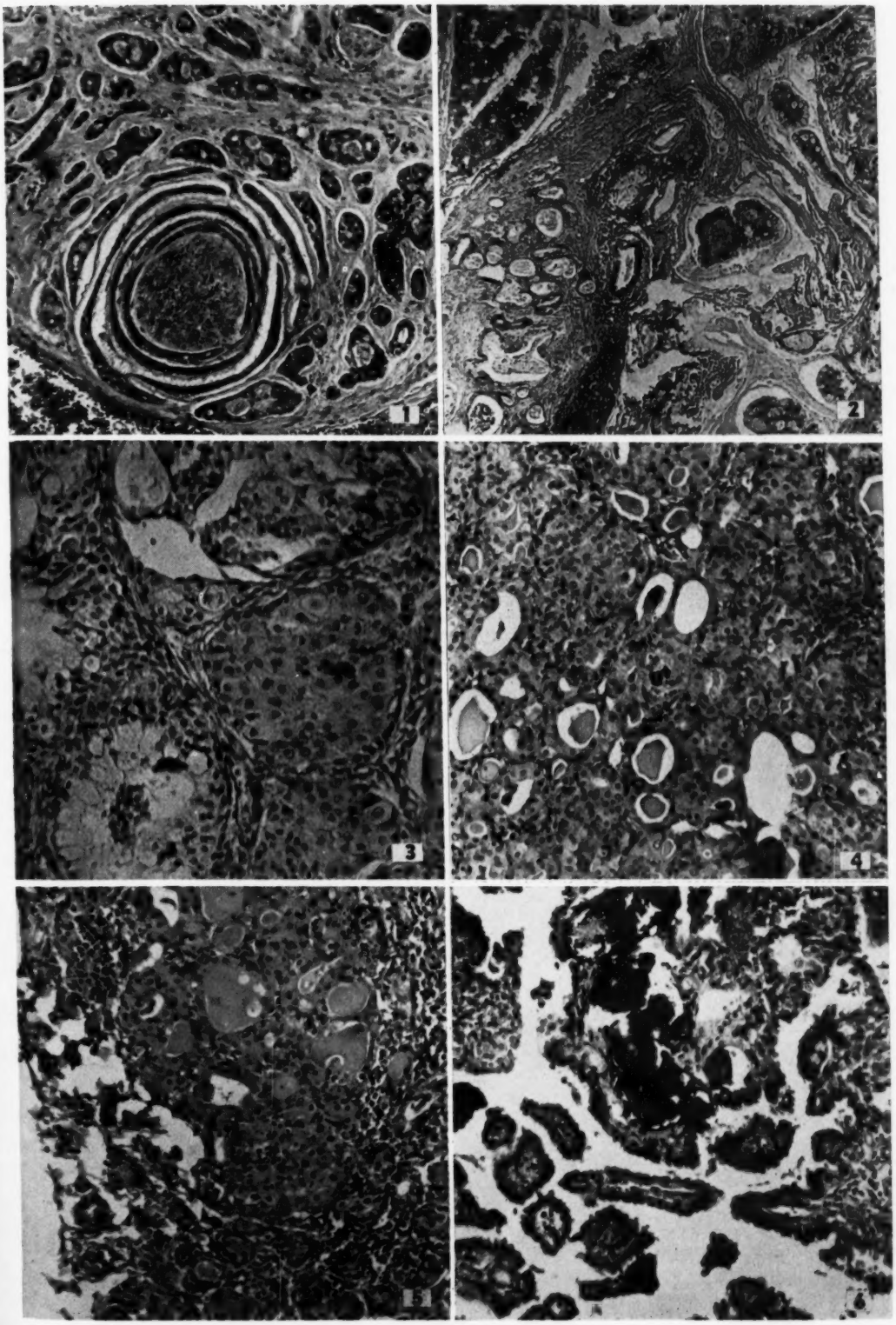


Fig. 1. Case 1. Adenoid cystic carcinoma, salivary type, showing perineural lymphatic invasion, a common finding.

Fig. 2. Case 2. Low power view of low grade mucoepidermoid carcinima of parotid.

Fig. 3. Case 2. Mucous and squamous components of mucoepidermoid carcinoma.

Fig. 4 and 5. Case 3. Chronic thyroiditis versus struma lymphomatosa.

Fig. 6. Case 4. Papillary carcinoma of thyroid, grade 1, with psammoma bodies.

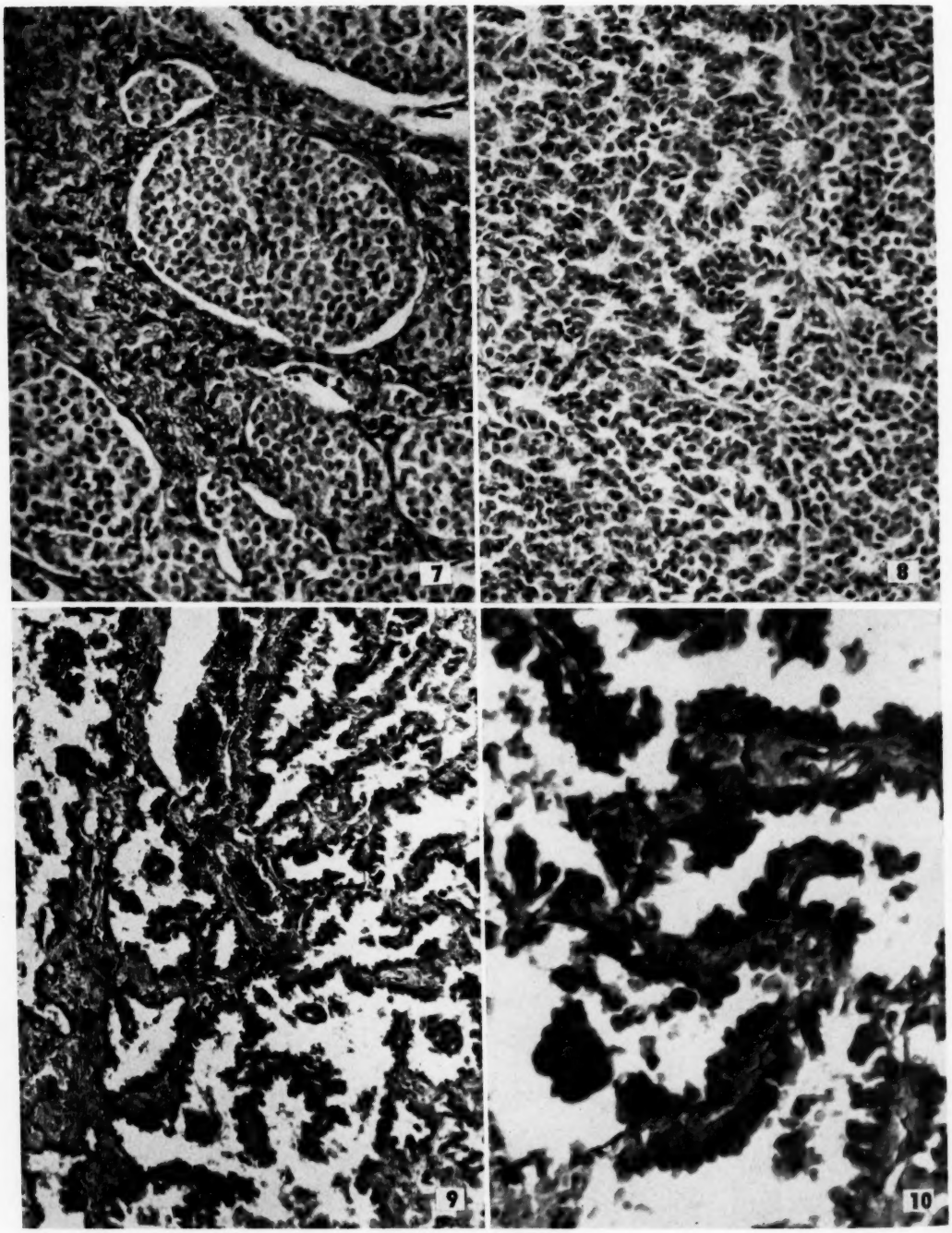
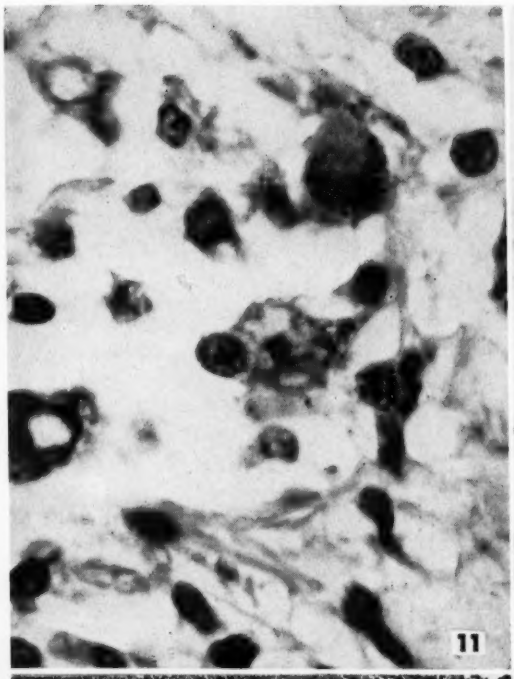
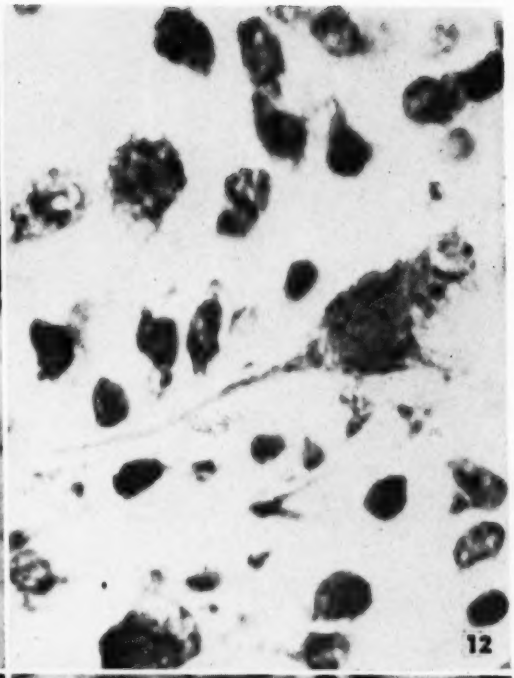



Fig. 7. Case 5. Malignant islet cell tumor of pancreas.
Fig. 8. Case 5. Subpleural metastasis of tumor shown in fig. 7. Note the structural similarity to so-called bronchial adenoma.

Fig. 9 and 10. Case 6. Low and high power views of terminal bronchiolar or alveolar cell carcinoma.



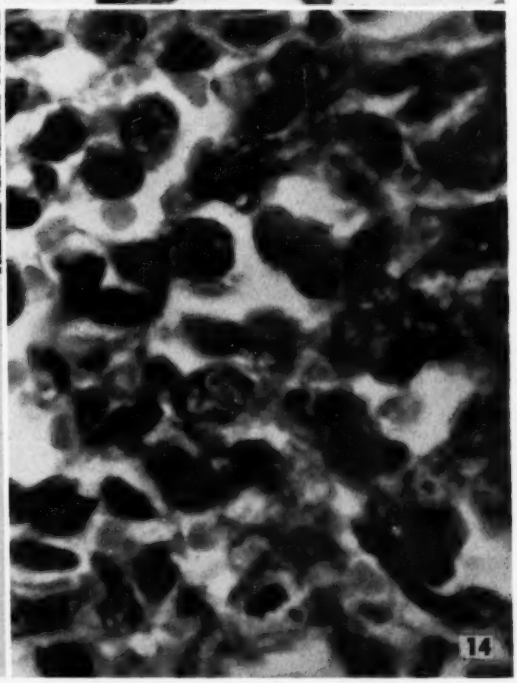



Fig. 11. Case 7. Pigmented ganglioma cells and smaller neurocytic elements of unusual pigmented ganglioneuroblastoma.

Fig. 12. Case 7. Bodian silver preparation showing "taproot" ganglion cell. The term is N. C. Foot's.

Fig. 13 and 14. Case 9. Low and high power views of malignant melanoma of gallbladder, metastatic.

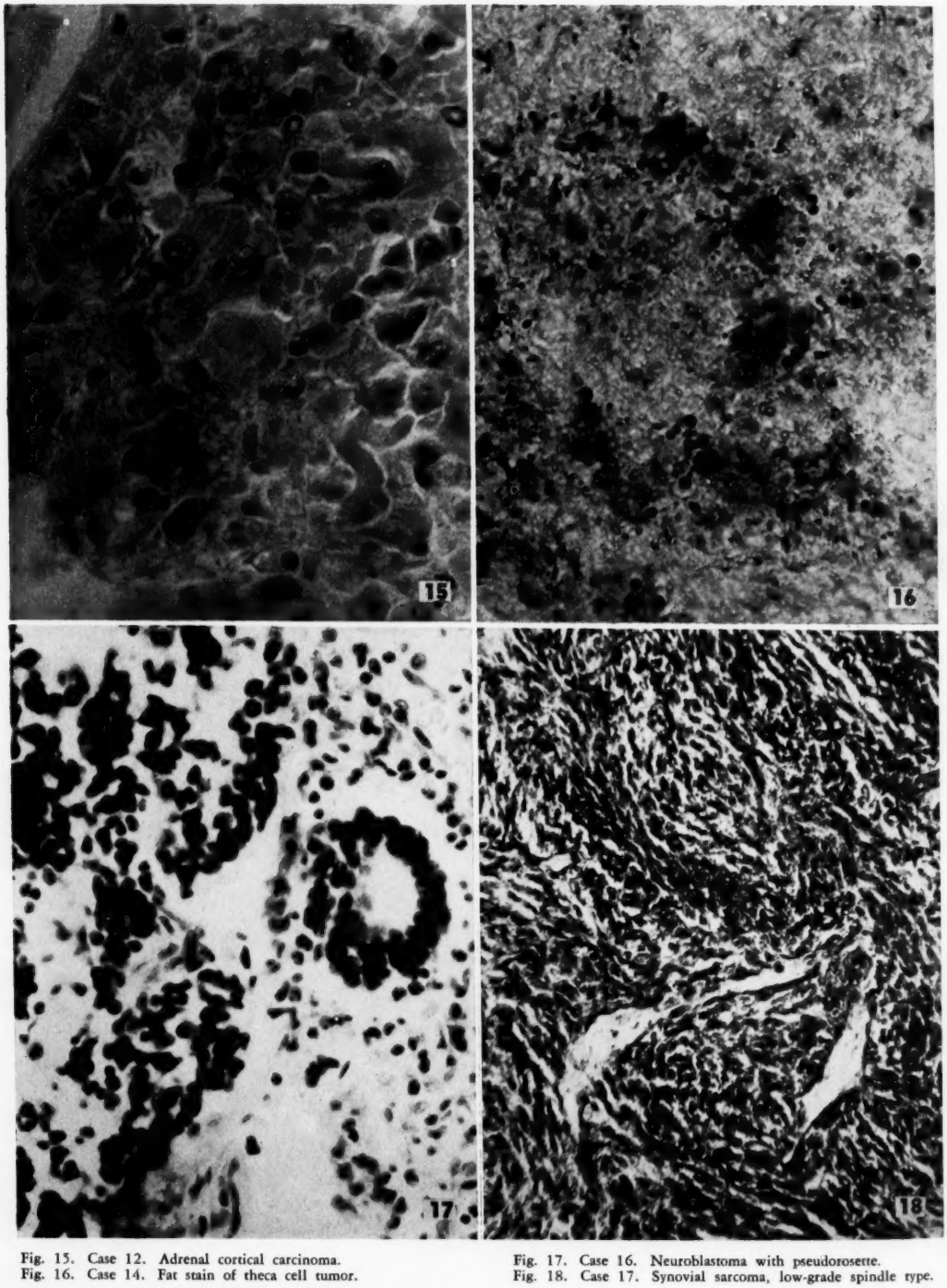



Fig. 15. Case 12. Adrenal cortical carcinoma.
Fig. 16. Case 14. Fat stain of theca cell tumor.

Fig. 17. Case 16. Neuroblastoma with pseudorosette.
Fig. 18. Case 17. Synovial sarcoma, low-grade spindle type.

died ten days after the operation from an obstructive ileus due to fibrinous intestinal adhesions. On autopsy, a tremendous liver was found weighing more than 3,000 Gm. and studded with large metastatic tumor nodules; metastases were found all over the peritoneum. The uterus contained numerous partly calcified leiomyomas, but none of these was considered grossly malignant, and it was not considered likely that a primary uterine sarcoma would metastasize to the liver, rather than to the lungs. Further exploration therefore was done, and a less than cherry-sized tumor was found in the wall of the ileum, which was ulcerated at the lumen and perforated into the peritoneum. This proved to be a leiomyosarcoma of low morphologic malignancy and was considered the primary tumor. The ulceration explained the previous episode of melena.

The third case is that of a 43 year old woman who had a history of only five days of abdominal pain, with a palpable abdominal mass and peritoneal irritation. On laparotomy large and small tumor masses were found throughout the peritoneum, one of which was intimately attached to a small intestinal loop, 21 cm. of which were resected. The tumor masses were twisted and hemorrhagic. A smaller, harder mass was attached to the uterus. The tumor presented as a cellular leiomyosarcoma of the ileum with spread throughout the peritoneum. The tumor was cellular, but mitotic figures were not too frequent. The uterine tumor was a benign subserous leiomyoma.

It is striking to note the difference in the clinical behavior of this tumor despite the similarity in structure. In the first case, there was a large tumor with a protracted clinical course over five years. In the second case the clinical symptoms were of only one month's duration, and the discrepancy between the very small primary intestinal tumor and the enormous metastatic liver was striking. In the third case, the clinical symptoms were of only five days' duration, obviously due to twisting and hemorrhagic infarction of tumor nodules, yet huge tumor masses were found on operation. In all cases, the morphologic structure of the tumor was similar despite the different biologic behavior.

Dr. Foote: Thank you, Dr. Lowbeer. Your case in which there were multiple uterine fibroids called to mind a case that I saw a good many years ago from the collection Dr. James Ewing had. He had gotten the material from Dr. Paul Steiner of Chicago, and the pattern of spread in this tumor was extremely peculiar. It had seeded the lungs in a diffuse way, and I do not believe anybody could have taken a photomicrograph of the uterus or the lungs in that case and sold it as a malignant tumor. It looked benign, strictly. Well, after all, those extraordinary events of benign looking yet metastasizing tumors do come up now and then. Once in a while a meningioma, for no particular reason, moves, and we have a few ordinarily nonmetastasizing basal cell epitheliomas that get about. Certain histologically benign looking giant cell tumors will disseminate. The so-called benign metastasizing struma of the thyroid is well known, but not entirely in this category. We have to be prepared to accept unusual performance from time to time.

Dr. Lloyd R. Hershberger, San Angelo: Is there merit to looking for mitotic figures in cellular smooth muscle tumors? When not present, may they be considered benign?

Dr. Foote: I am always wary about any question dealing with mitoses. If I said that they were important, somebody immediately could bring out case after case of uterine myomas, particularly during pregnancy, in which there were lots of mitoses and a strictly benign course. If I took the other point of view, you could bring out exceptions that would debunk it. I would say that in a tumor such as we have here the mitotic rate might be given consideration. If it were very, very low I would not be at all influenced in the diagnosis. If it were very, very high I would just be surprised. I do not believe that you can settle important questions as to prognosis solely by the rule of mitosis in spite of the many papers that our valued friend, Albert Casey, wrote on the subject. One can draw graphs and curves with mitoses counts and get a nice correlation, but one also can correlate about a dozen other attributes and come out with the same thing. I do not disrespect mitoses, nor do I look upon them as a deity.

Audience Member: Are you surprised that there is not a single melanoma diagnosis from the audience on this case, or do you think there is no similarity between leiomyosarcomas and melanomas?

Dr. Foote: I would not discount melanoma 100 per cent under any condition, because the structural range of the lesions is too extreme, but I would not have given melanoma any real notice here. This thing just looks like a leiomyosarcoma, although I must admit I could not tell anybody precisely how leiomyosarcomas look. They've got a face and a figure just like boys and girls have. I cannot really describe a leiomyosarcoma to suit myself, let alone anybody else, but almost everybody thought that this was a smooth muscle lesion, and that is a better answer, I think, than I could promote.

Audience Member: We happened to have one like this in the North Texas Society of Pathologists, and

it later turned out that the patient had had a melanoma of the skin.

## CASE 11

*Diagnosis.*—Wilms's tumor.

*Contributor.*—Dr. H. J. Schattenberg, Clinical Laboratory, San Antonio.

*Specimen.*—Tumor mass.

*History.*—The patient was a white infant, aged 2 months, who died suddenly. At autopsy an ovoid tumor mass measuring 4 by 6 cm. was found within the abdomen.

*Submitted Diagnoses.*—Embryonal renal tumor; hypernephroma; islet cell tumor of pancreas; granulosa cell tumor of ovary.

Dr. Foote: I wonder if Dr. Schattenberg has available any supplemental information on the gross pathology?

Dr. H. J. Schattenberg, San Antonio: When I was asked whether there was any connection between this tumor and the kidney, all I could say was that it was free and independent of the kidney and lay on the upper pole of the kidney, pressing the kidney in that locality, but free and independent of it. It seems to have been in place of the adrenal gland, as no adrenal could be found on that child.

Dr. Foote: That clarifies that this was an extrarenal tumor. I do not know how, in this case, we are going to be able to avoid making the diagnosis that almost everyone has made. I was not really overwhelmed when I learned that this tumor was extrarenal. I have seen 1 tumor in a youngster that was classical of embryonal adenomyosarcoma, and it lay above the level of the diaphragm. This was a case of Dr. R. J. Shafer of Corning, N. Y. Autopsy was performed, and both kidneys were free. We were forced to regard it as an ectopic Wilms's tumor. I had 1 other autopsy on an adult who had a distinctly extrarenal Wilms's tumor.

I did not select this case as a problem of histologic diagnosis, but did think the fact of the tumor's ectopic position was interesting. Moreover, in recent years information of interest dealing with treatment and prognosis has accumulated, and pathologists always should be interested in correlating structure with course.

In this connection I wrote to Dr. Sidney Farber of Boston, knowing that he had seen a great deal of critical material of this sort at the Children's Hospital, and had seen all the cases that had been reported by Ladd and White and other clinicians from the Boston area. He sent back the interesting comment that he did find one histologic principle of importance in prognosis, namely, that in the favorable cases he seldom was able to demonstrate striations, and hence the presence of striations to him was ominous in the Wilms's tumor group. That experience is not universal. In the Mayo Clinic material, out of some 40-odd cases, 43 I believe, striations were found in 20. This is the Dockerty and others report. They were unable to find striations a factor in their survivals 6 or 7 in number. There is something about the Boston material that is rather prepossessing. As long ago as 1941, the group there reported on 60 cases. Eighteen patients were still living in 1941; 14 of the 18 were regarded as cures and 3 were thought to have a hopeful prognosis. Ordinarily the reported mortality rate from the disease ranges up to 90 per cent.

In the 1941 paper the Boston authors pointed out an important point in prognosis, the factor of age. Fifty per cent of the cures were in infants 1 year old or less. In that connection I asked Dr. Farber if he thought that age influence could be explained on size of the tumor. I had reasoned that these tumors were perhaps congenital and that if they were operated on when the patient was 1 or 2 years of age they would be smaller, more easily operable, and have less time in which to become bulky and yield metastases. Dr. Farber did not entirely eliminate that, but he thought that in the favorable results the change in surgical approach was highly important, probably instrumental—the adoption of the transperitoneal method of surgical removal. That apparently has been a policy with the Boston group for a long time, and in one of the Cabot case discussions just a few years ago Dr. Landman traced the cure rate by decades. The over-all cure rate, irrespective of time, was 35 per cent, amazingly high for Wilms's tumors. That is something that cannot be ignored. From 1914 to 1930, they had 26 cases and 4 cures; from 1931 to 1939, there were 30 cases with 10 cures; from 1940 to 1947, there were 37 cases with 19 cures. That later material coincides with the standardization of the immediate transperitoneal surgical attack without preoperative irradiation. So I think that that Boston report is something to bear in mind when we are faced with the problem of treatment of Wilms's tumor. The age factor seems to be important and apparently the method of surgical approach. There will be residual debate as to the place of irradiation as an aid in cure.

## CASE 12

*Diagnosis.*—Adrenal cortical carcinoma.

*Contributor.*—Dr. L. J. Manhoff, Jr., Robert B. Green Hospital, San Antonio.

*Specimen.*—Adrenal cortex.

*History.*—This Latin-American woman, aged 28, had had symptoms of amenorrhea, obesity, and marked hirsutism for four years. Urinary 17-ketosteroid was 400 mg. in twenty-four hours. Perirenal air insufflation exhibited a large left adrenal mass which at operation measured 10 cm. in diameter and weighed 300 Gm. It was moderately soft and

globular, and had a smooth capsule. The cut surface presented a homogeneous reddish brown surface with scattered areas of hemorrhage.

*Submitted Diagnoses.*—Adrenal cortical adenoma; adrenal cortical carcinoma; pheochromocytoma; chromaffinoma; sympathicogonioma.

Dr. Foote: The appearance of this patient at the peak of her disease, and the alteration of her facies and body postoperatively are really impressive. I would like to make one point; that is, when somebody brings you an ovarian slide and you feel a quick compulsion to make a diagnosis of adrenal cortical carcinoma, do not take the bait too fast. It may be a pheochromocytoma. It is within our rights to ask for information, and in the absence of information it is possible to confuse such obviously chemically different tumors as the pheochromocytoma and adrenal cortical carcinoma. If you get out a dozen tumors of those two orders and look those slides over one after another, you cannot but be impressed with the extremely broad structural range within those groups. If you do not find chromaffin cells in those tumors, there are many, I believe, with which one would stumble.

I believe this tumor is malignant. The reason is that most people who have lesions of the adrenal that look like this are dead. The fact that there may be some alive only means that they either were lucky, had good treatment, or both. This type of tumor is commonly fatal when it occurs in young people, 30-odd out of 70 cases having run a malignant course, and it is said to be even more aggressive in older people. So I would say without hesitation and irrespective of follow-up and future course that this was an adrenal cortical carcinoma. The fact that something which was called a carcinoma has not killed the patient does not mean that it was not a carcinoma. Otherwise, there would never be a cure of cancer. The steroid chemists would have a lot of fun with this case. They had a lot of data.

I suspected that someone would want to particularize upon which cell of adrenal cortex was concerned in this woman's trouble. There have been some efforts, of course, in the past to pin down functionability on certain areas of adrenal cortex, and whether or not Wilkins' work is still acceptable to qualified critics, it is rather interesting. The evidence seems to make a plausible story of the probability that the reticular zone is concerned with the production of the androgenic hormones, but I do not think the case is proved. This work would bear out the statement that Dr. Hollander made on malignant tumor versus adrenal hyperplasia, differentiating benign and malignant presentations. Wilkins, Fleischmann, and Howard had 1 extraordinary patient in whom they identified almost all of the adrenal cortex as being made up of cells of the reticularis, to the apparent total extinction of the glomerulosa and the fasciculate layers, and the patient interestingly died of Addison's disease.

I would like to show another rather extravagant case of this sort. Here is the patient; I do not believe that the clinical photographs require any extensive elaboration. We notice male escutcheon, slightly protuberant abdomen, a little thickening of the thighs and hips, a little breast atrophy, and an enlarged clitoris. I believe there is another clinical photo that should be shown with this. The patient was a Powers model and a person of wonderful figure and grace, and the alterations are terrific. She had a primary tumor of the adrenal. She had an excretion pattern which I have not seen completely reported, but she had perfectly enormous quantities of androgenic hormone. She also had a decreased sugar tolerance, and was at a diabetic range. The surgical specimen of adrenal cortical carcinoma looks much like the description given for the conference case. At the time of autopsy the tumor was growing into the inferior vena cava. After the surgical operation there was a temporary decline in the level of the patient's hormone output, but it rapidly rose again to assume even higher levels, and about eleven months after her operation, she died suddenly. An autopsy was performed, and her death had been due to the rupture of a liver metastasis with a massive hemorrhage. She also had pulmonary metastases, local recurrence, and tumor still in the inferior vena cava.

## CASE 13

*Diagnosis.*—Mixed mesodermal tumor.

*Contributor.*—Dr. J. M. Moore, Santa Rosa Hospital, San Antonio.

*Specimen.*—Uterus.

*History.*—This patient was a woman, aged 55, with a greatly enlarged uterus measuring 18 by 14 by 10 cm. There were multiple fibroids in the walls with a large degenerated mass in the fundus. This was degenerated over the surface, soft in consistency, and appeared to be of fatty tissue.

*Submitted Diagnoses.*—Endometrial stromal sarcoma; leiomyosarcoma; rhabdomyosarcoma; sarcoma, unclassified; mixed mesodermal tumor; carcinoma of corpus; stromal adenomyosis (endometriosis); carcinosarcoma; metastatic carcinoma; malignant mesenchymoma.

Dr. Foote: When I first saw these slides, I thought that we would have an opportunity to develop a more definite diagnosis than I think can be squeezed out of this with certainty. I was hoping we could turn up structural qualities that would justify a diagnosis of a mesodermal mixed tumor. I see that four have been optimistic about that. I would like to ask if anybody saw any demonstrable epithelium or a striation in this case, because without something be-

sides what we have seen in the slide distributed, I do not believe we can work that into the picture. Although my auspices and the guiding light tell me that this is a mesodermal mixed tumor, those authorities have been wrong so many times I can't trust them. Also, I had no way of settling whether or not this is an endometrial sarcoma or a leiomyoscarcoma. I would like to hear some comments from the floor on this subject.

Dr. Leo Lowbeer of Tulsa asks, "Have you observed collagen producing sarcomas of the myometrium?" I would answer that immediately by saying that I think the common myoma frequently puts down a great deal of collagen, and the same is true of leiomyosarcomas. Dr. Lowbeer states that he had a case of a large fibroblastic sarcoma of the uterus, probably originating within a leiomyoma, with an unusual reaction of neutrophil and eosinophil leukocytes and the formation of a large pseudo-abscess. The tumor was 30 cm. in diameter. That must be close to a record. I do not see anything at variance with possibilities in the report of a case like that. A pure fibrosarcoma of the uterus is no doubt a rare lesion, and I think it is very difficult to establish acceptably rigid diagnostic criteria for the differentiation.

Dr. William W. Tribby, Memphis, Tenn.: I wonder if Dr. Moore could give a little more detailed information concerning this case, especially the large degenerated mass in the fundus? I wonder if that might have been attached to the exterior surface of the fundus or whether it grew into the interior of the uterus. This tumor looked like a liposarcoma to me, and I wondered if it might have arisen from some adherent fat in the fundal portion of the uterus, attached to the peritoneum.

Dr. J. M. Moore, San Antonio: The tumor was inside the fundal cavity, attached up near the top of the fundus, and it was a very large tumor. Besides this, there were multiple nodular fibroids in the musculature.

Dr. Foote: That would dampen our liposarcomatous ardor a bit, wouldn't it? And then it would make it still difficult to decide between these and other possibilities. I would despair of being able to make a categorical diagnosis in a case of this sort.

## CASE 14

*Diagnosis.*—Theca cell tumor.

*Contributor.*—Dr. L. J. Manhoff, Jr., Robert B. Green Hospital, San Antonio.

*Specimen.*—Ovary.

*History.*—This Latin-American woman, aged 24, was admitted to the hospital because of abdominal pain of forty-eight hours' duration. Sterile fluid was aspirated from the abdominal cavity. A large abdominal mass palpated in the lower part of the abdomen was found at operation to be an ovarian tumor on a short pedicle. The tumor measured 16 by 10 by 10 cm., was well encapsulated, and had a smooth surface. On cut section it exhibited a somewhat soft, yellowish, homogeneous tissue in which there were numerous areas of hemorrhage and cystic degeneration.

*Submitted Diagnoses.*—Thecoma; fibroma; granulosa cell tumor; luteoma; arrhenoblastoma; hemangiopericytoma; liposarcoma.

Dr. Foote: Here we have a problem of selection. The gross tumor was circumscribed and encapsulated, and microscopic section showed cellular areas. I think all of the submitted diagnoses could be anticipated because of the structural gradients that overlap one with another, but I would not have invested any money in liposarcoma and hemangiopericytoma, and I would have passed over lightly on arrhenoblastoma.

The thing actually dissolves down to a point of how much theca, how much granulosa, you think this tumor has in it, and is it or is it not luteinized. Well, what means can be employed to clarify that? Everyone will ask immediately for a fat stain. It was positive. It was taken from a nondegenerated area of the tumor. What does the fat mean? I don't know. It presumably would be doubly refractile material, and if chemically analyzed no doubt would come out high in cholesterol and cholesterol esters. Do we make a diagnosis of theca cell tumor on that basis? Most people do. Do we eliminate the diagnosis of theca granulosa? No, you can't. They are almost inextricable. Can we partition it in terms of degree? Why, anybody can make his own guess about that.

Perhaps if you are a Traut-Marchetti fan you would like to have a reticulin stain on this, inasmuch as Traut and Marchetti and others since have thought that reticulin staining was of use in these cases to detect the amount of theca versus granulosa element. They maintain that theca cells typically are stained in the Graffian follicle and in tumors alleged to be of that origin are neatly wrapped up with very, very tender little morsels of thin and delicate reticulin—"l-i-n." Dr. A. P. Stout insists on that; I used to call that "reticulum" before I was better educated. If we had that stain, I think that we probably would see what is usually predicted for it.

The thing that would interest me more would be whether or not you can tell if the tumor is luteinized. Large accumulations of fatty material sometimes are considered synonymous with luteinization, but I don't think that is by any means necessarily so, because you do not have any specific chemical reaction or test on the material itself to establish it, and accumulation of fat in the cells does not necessarily mean that it is lutein material. Unless we had some urinary excretion studies which showed a pregnandiol output that was elevated; unless we had some evidence of

luteinization from the endometrium, I do not know how we could ever say that any particular tumor is or is not luteinized. Certainly some of the theca granulosa tumors are capable of luteinization.

There is a well reported luteoma in Novak's book, and some years back Twombly described an interesting case from which we saw the material at Memorial. The patient was a young girl who had an ovarian tumor which had theca and granulosa cell elements in it and a lot of cells that were literally stuffed with fat. She had an increased pregnandiol output and also a uterine curettage which showed a decidual reaction. She was a young girl from a very religious family. Her habits were absolutely impeccable, unimpeachable, and her hymen was intact, so I suppose the hormone from her ovarian tumor was giving this decidual reaction. We signed this out as a luteinized theca-granulosa cell tumor.

Most people think when you have fibromatous looking tumors of the ovary, well preserved, in which you get a positive fat stain, you have a theca cell tumor. That is good enough for me. I do not worry much about those things. The only thing that worries me about those tumors is the fact that in discussions at various meetings clinicians repeatedly refer to the fact that these tumors are not strictly benign tumors as sometimes thought. At a recent conclave one of the commentors stated that if these cases are followed for long periods, up to 25 per cent of these tumors will recur, and in some instances cause death.

## CASE 15

*Diagnosis.*—Mucinous adenocarcinoma.

*Contributor.*—Col. C. J. Lind, Brooke Army Hospital, Fort Sam Houston.

*Specimen.*—Urinary bladder.

*History.*—This white man at 48 years of age underwent an endourethral resection of the prostate for hypertrophy. Two years later he underwent a segmental bladder resection for tumor.

*Submitted Diagnoses.*—Mucinous adenocarcinoma; adenocarcinoma of urachal origin; adenocarcinoma; carcinoma of prostate; colloid carcinoma; mucoid carcinoma (probably from colon); mucus-producing carcinoma (Cowper's gland).

Dr. Foote: This case was selected more as a rarity than as a diagnostic problem. Almost everybody has made a diagnosis of adenocarcinoma, of one place or another. I think the specific designation of origin from urachus is plausible, to be sure, but you cannot prove this one way or another because we do not have exact anatomic tracing to the urachus. Yet the bulk of anatomic evidence does favor a urachal site. The segmental resection was made at the central dome of the bladder, and at autopsy the tumor was recurrent and involving bladder wall. The man had bulky intra-abdominal disease, apparently lying in the midline extending from the bladder. The wall was diffusely involved; there was a little polypoid excrescence poking its head through into the lumen of the bladder. Microscopically the tumor was a mucoid adenocarcinoma and was positive by mucicarmine stain. This case, as far as I am concerned, requires no particular discussion.

Dr. Leo Lowbeer, Tulsa, Okla.: Have you ever seen a primary cancer of the bladder with a structure like that in the infiltrating signet ring cell carcinoma of stomach or rectum? We observed a case in which such a tumor completely infiltrated the entire bladder wall and to a lesser extent the sigmoid, rectum, and all adjoining structures including the anterior muscles of the thigh. The predominant symptoms were bladder symptoms.

Dr. Foote: Yes, I think this one looks just like many tumors of the stomach and rectum. I could not tell this from any gastrointestinal cancer. Dr. Lowbeer, if you had a primary complex in either stomach or intestine, I think I would put that as a first choice as primary with peculiar secondary involvement of the bladder. Lacking that, I would not be able to put it on an intestinal basis.

Most of the mucous cell adenocarcinomas of the bladder are reputed to be of urachal origin, to be sure. That is not an absolute requirement, however. We have 2 cases that we think probably are primary in bladder epithelium. Both of these originated in the bladder base, in the trigonal region. I do not mean that that eliminates urachal origin, but in neither case could urachal structures be detected, and also the ability of the bladder mucosa to undergo extraordinary metaplasia has been known for many years. As a matter of fact, Zuckerkandl wrote about that a long time ago. Intestinal metaplasia from time to time occurs. I think almost everybody sees it once in a great while, and the capacity for remarkable metaplasia of transitional epithelium is reflected even as high up as the kidney pelvis. Remember, a few years ago Dr. N. C. Foot reported what he called pyelitis cystica with intestinal metaplasia; and so mucous cell cancer does not have to be of urachal origin in the bladder.

## CASE 16

*Diagnosis.*—Neuroblastoma.

*Contributor.*—Dr. L. J. Manhoff, Jr., Robert B. Green Hospital, San Antonio.

*Specimen.*—Presacral mass.

*History.*—This white boy, aged 6 months, was admitted to the hospital because of urinary retention of three days' duration. The patient died during anesthesia for cystoscopy. At autopsy there was a retroperitoneal mass attached to the anterior surface of the sacrum measuring about 6 by 4 by 3 cm. The mass invaded the sacrum and the extradural space.

*Submitted Diagnoses.*—Neuroblastoma; sympathicoblastoma (sympathogonioma); teratoma (undifferentiated, ma-

lignant sacrococcygeal, with papillary adenocarcinoma); glioblastoma; nephroblastoma; malignant tumor.

Dr. Foote: A presacral mass—infant of 6 months—without looking at the slide, with that amount of information, a highly probable diagnosis is neuroblastoma because hardly anything else would produce that, assuming no bone involvement. Almost everyone favors a malignant tumor of the nervous system. I did not have a diagnostic problem particularly in mind when this case was selected, realizing that it would be readily recognized; but there were a few facts about the disease type that I think are worthy of passing comment.

This tumor had nice pseudorosettes and I say pseudorosette because in the middle of this structure we have what looks like a fibrillary matrix. A true rosette, according to the J. Homer Wright version, should not have anything in the middle, and this does. There were no neurofibrils found in that area. They are said to occur now and then, but they must be very difficult to demonstrate.

About the only thing in these tumors that is subject to discussion is terminology. I notice here that most people have selected neuroblastoma, and in so doing I think they follow the suggestions that Dr. A. P. Stout has made in tumors of this sort. He refers to all of these lesions, irrespective of minor differential features, as neuroblastoma, whereas other pathologists would perhaps go back and favor (on account of the pseudorosettes in here) a diagnosis of sympathicogonioma. It does not make much difference from the prognostic viewpoint, or the response to treatment, although once in a while you see some statement by somebody that the degree of differentiation of these tumors is extremely important. I could not see much importance in it unless it were a benign tumor. These sympathicogoniomas, sympathicoblastomas, and neuroblastomas are all highly malignant tumors. We have a few, a very few, that we characterize as ganglioneuroblastomas. Not enough of those are recorded to enable coherent comments.

Briefly, I think it worth while to point out that the general attitude toward neuroblastoma is one of near total hopelessness. The record of the disease is not quite that bad, but is bad enough. In the recent Hunterian lecture given by Dr. Ralph Philips are some interesting data on the disease. He documented the material that he studied at Memorial Hospital and analyzed reports from other sources. He had a total study group of about 80-odd cases, of which 58 were treated, and he had a number of long-term survivals. It looks as if there will be a significant number of complete cures. There are 5 patients treated and alive without evidence of disease after from six to thirteen years; there is another five year case and 2 more followed three years without any evidence of disease. In the control of these patients there has been a combination of treatment methods, of course, so that you cannot accurately evaluate any single one. Suffice it to say that surgical, irradiation, and other forms of treatment have been employed, including certain chemotherapeutic agents.

One of the cases in particular interested me. I saw sections from it many years ago. There were widespread metastases, but the case proved to be a clean example of spontaneous regression. So we cannot leave neuroblastoma without pointing out that it is not an utterly hopeless disease, and important enough in itself would be the fact that most of those tumors are nicely radiosensitive. In the widely disseminated case, important palliation may be secured from the judicious use of external irradiation therapy, alone or combined with certain chemotherapeutic agents.

## CASE 17

*Diagnosis.*—Malignant synovioma.

*Contributor.*—Dr. A. O. Severance, Baptist Memorial Hospital, San Antonio.

*Specimen.*—Lung.

*History.*—This white woman, aged 26, complained of a tumor of the left wrist which had been present for eight years and which had been excised frequently with recurrences over a period of a year and a half. A chest roentgenogram revealed several rounded shadows in the right lung.

*Submitted Diagnoses.*—Synovioma, malignant; fibrosarcoma; myosarcoma; malignant neurilemoma; malignant Schwannoma; reticulum cell sarcoma; neurofibroma.

Dr. Foote: We have a documentation of the case down to the point of a pneumonectomy. Remember that this patient had had the tumor for about eight years before intervention, so it had marked itself already as a sluggish grower. A recurrence occurred in 1952 and again in 1953. Throughout this period I believe this tumor was variously regarded as a low-grade fibrosarcoma or a synovioma, and just how to make a clean distinction is worth some discussion. I should think that a diagnosis of either low-grade fibrosarcoma or low-grade synovioma would be satisfactory in this situation, at least as far as practical matters are concerned, because mode of attack would not be substantially different irrespective of diagnosis. The reticulin stain in this case, according to Dr. A. P. Stout's teachings, was more in line with a synovial tumor than a fibrosarcoma because a great many tumor cells were contiguous and not intersected or ensheathed by reticulin fibers, which he points out are characteristically present about the cells of low-grade fibrosarcoma. That is all well and good. When fibrous tissue tumors begin to assume a more malignant aspect, these traditional reticulin stain determinations break down. Actually, when you need

it most, it does not help a great deal. Perhaps at the histologic level of this case it is contributory and of aid. I made a diagnosis of synovioma on this case, and Major Sproat, after a long search, found these areas that seem to represent at least a pseudoepithelial growth. None of these structures could be found in any of the material from the local lesion. And so it seems correct to refer to this as a low-grade synovioma.

There was a large vote in favor of the malignant synovioma, and fibrosarcoma was in second position. If those positions had been reversed, I would not have been startled. When the synovial tumors do not reveal their nature by forming epithelial-like areas, they are extremely difficult to diagnose with certainty. I suppose all of our diagnoses of low-grade synovioma and fibrosarcoma are subject to some error, no matter who makes the diagnoses. Here are other lantern slides of a case that we had some years ago—one of these low-grade, rather orderly looking synovial tumors—with the reticulin stain more indicative of the structural traits of synovial cells than the fibrocytic series. There is no problem in the diagnosis of synovioma when you have the necessary structural features such as classical epithelial looking synovioma, partly spindle but partly growing almost like carcinoma.

The one point worth mentioning in discussing synoviomas is that those tumors that grow principally as spindle cell and rather orderly looking ones are uniformly of slow evolution and apt to run a long period of years before metastasis or fatal termination. They are highly prone to local recurrence, but in some cases you can have a long interval between recurrences. The danger point in these synoviomas is in the third and fourth year. We have a lot of recurrences and metastases that take place about that stage when it looks like the case is going to run a clean course.

Dr. A. O. Severance, San Antonio: I can give the follow-up if you wish. This patient was operated on in January of this year and the right lung was removed. The lung was filled with numerous nodules and some of them were metastatic nodules such as you saw and which we felt were malignant synovioma or synovial sarcoma. There were other nodules, equally numerous, of varying size, with caseous centers which were granulomas of one form or another, but we thought maybe they could be considered healed tuberculoma or healed tuberculosis. This patient had this operation because she was pregnant. She wanted to live long enough to see the child born, and there was some possibility that she might live a little longer if the pneumonectomy was done. At the time of the operation, in the left lung there was a nodule, presumably metastatic, but after the right lung was removed there was the question of whether that nodule was granuloma or metastatic tumor.

Time has gone on. The baby is now 6 months old, the woman is still alive, but not doing too well. Fluid accumulated in the left lung, and at the present time there are 2 nodules in the left lung which were not present at the time of pneumonectomy. The nodule that was present at pneumonectomy has not increased in size, but these 2 other nodules have appeared and are growing and are presumably metastatic. The patient does her own housework, takes care of her own baby, and aside from the pain and discomfort, the necessity for removing the fluid from that left lung, she is getting along moderately well. She has to have Demerol for relief of pain, and her doctor thinks she is slowly on the downhill course.

## CASE 18

*Diagnosis.*—Embryonal rhabdomyosarcoma.

*Contributor.*—Col. C. J. Farinacci, Fourth Army Area Medical Laboratory, Fort Sam Houston.

*Specimen.*—Mass from thigh.

*History.*—This white girl, aged 11 months, was admitted to the hospital with the history of three months' development of a tumor of the left thigh without other symptoms. At operation a specimen measuring 12 by 3 cm. was removed. There was a filmy capsule beneath which the tumor was firm, white, and homogeneous, but it could be broken apart easily. Roentgen-ray examination revealed no sign of bone involvement.

*Submitted Diagnoses.*—Liposarcoma; mesenchymoma; miscellaneous sarcoma; embryonal sarcoma (sarcoma botryoides); synovioma; hemangioendothelioma; neuroblastoma; neuroepithelioma.

Dr. Foote: I am startled with liposarcoma. That is a vanishingly rare tumor in infancy. Scarcely a good straight-forward case can be made out of liposarcoma at that age. The youngest patient that I have ever seen reported was aged 3 years. That case was a phony. The only reason it ever got reported as a liposarcoma was that somebody talked Dr. James Ewing into calling it that, and he later relented on that diagnosis. The case got published, though. I would be exceedingly afraid of liposarcoma and would want a really typical one before suggesting that diagnosis in an extremely young person.

I thought about mesenchymoma. They are getting popular although until a few years ago we never saw that diagnosis. Since Dr. A. P. Stout popularized it, it has caught on like old time scarlet fever and everybody has a heap of mesenchymomas. It looks like this tumor is getting more abundant than Dr. Stout had planned. His 8 cases of mesenchymoma he reported and considered malignant just a few years ago.

I was much pleased to see five diagnoses of embryonal rhabdomyosarcoma. People around New York are telling us that we are making too many diagnoses

of embryonal rhabdomyosarcoma. They don't know what they are talking about. We do not make enough of them, I'm afraid. We miss that diagnosis sometimes when we ought to make it, actually, but it is going to be hard to sell in this case, I admit. I thought neuroblastoma might get a little bit heavier treatment than it did, but only one person mentioned that. I notice that a good many went for various forms of sarcoma, including Schwannian, and botryoides, and so forth. The botryoid I would not join because if we do not confine that diagnosis to areas such as the base of the bladder, the cervix, and the prostate, in very young people, we have no syndrome of sarcoma botryoides, and by expansion we lose the identity of the disease.

Major Harry Sproat, Fort Sam Houston: This case had the onset of symptoms with the enlargement of the thigh in October, 1952. It first came to medical attention in January, 1953, at which time a biopsy was done. In February the child was subjected to exploratory surgery and a large tumor mass removed from the left thigh, specimen of which was distributed as part of the material to the attending pathologists. After the surgical removal, irradiation was given, but the child progressed steadily downhill and died in October, 1953, exactly one year following the onset of symptoms. At autopsy there was a large retroperitoneal tumor mass which extended from under Poupart's ligament on the affected side and involved all the organs, including the bladder, ovary, the gastrointestinal tract, pancreas, kidney, and adrenal. The kidney on the left side, which was the affected side, revealed the picture which you see on the left.

Dr. Foote: Our fat stain was positive. That, of course, would support the liposarcoma diagnosis, but on the other hand, fat staining for the diagnosis of liposarcoma is largely a snare and a delusion. When you need a fat stain to aid in the diagnosis of liposarcoma, it is usually negative. If it is positive, you don't need it. I think that this positive fat stain is a result of degenerative processes within the cell. You can get fat in almost any tumor that is not in good shape. In the autopsy material the cells revealed a distinctly acidophilic trend in the cytoplasm and what I would take to be the effort at forming small strap cells. Very unconvincing, to be sure, but I would attach some significance to the rather marked acidophilia, because that would be rather unexpected in such tumors as a neuroblastoma. To make a long story shorter, I could not find any striations in this tumor no matter how hard I tried, but I talked with someone yesterday who says that he saw them. Is he here today?

Col. C. J. Farinacci, Fort Sam Houston: I saw them.

Dr. Foote: Can we count on that now? You are the chief support for our diagnosis.

Col. H. A. Van Auken, Madigan Army Hospital, Tacoma, Wash.: I saw them also.

Dr Foote: Thank you, Colonel, you are sent from the angels above.

Dr. John B. Frerichs, San Francisco: I found striations in this also with the phase microscope. I could not satisfy myself that they were really there with ordinary light, but with phase microscopy they seemed right in character and spacing to be pretty good striations.

Dr. Foote: Well, a stroke for modern methods of microscopy! I am glad you mentioned that. It emphasizes the difficulty of getting provable evidence in support of a diagnosis of this disease. One reason around New York they say we are making too many diagnoses of embryonal rhabdomyosarcoma is that our critics cannot find these striations. We often cannot either, but we make the diagnosis anyway, and if you put the material together and analyze it from the clinical viewpoint and the pathologic, it makes a fairly characteristic disease pattern. I think that it ought to be a standardized diagnosis and that it is as predictably a disease entity as was predicted in 1910 by J. Homer Wright for neuroblastoma. Think how many years it took before neuroblastoma diagnoses were uniformly made. Time moves slowly in these issues.

We are approaching our declared time limit, and I want to refer for further discussion of this type of rhabdomyosarcoma to the *Cancer* article written by Stobbe and Dargeon about September, 1950, which gives a lucid account of the disease. (Applause.)

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Memorial Hospital.

## Research on Insecticide Poisonings

The mechanism of action of anticholinesterase insecticides and the therapy of poisoning resulting from excessive exposure is being investigated by the Louisiana State University School of Medicine's Department of Pathology. The widespread use of Parathion and TEPP (tetraethyl pyrophosphate) in agriculture, industry, and the home is causing an increased number of accidental exposures. Information on such cases, both clinical observations and autopsy studies on fatal cases, is needed for research. The department is prepared to give recommendations as to treatment for surviving patients and also will send, at its own expense, investigative teams to any part of the South. Collect telephone calls to report cases and request information will be accepted at any time, day or night, by Dr. Stanley Durlacher, New Orleans, Franklin 4141.

# CASE REPORTS

## LEIOMYOSARCOMA OF THE ESOPHAGUS

OSCAR CREECH, JR., M. D.; ROBERT C. OVERTON, M. D.; and ETHEL ERICKSON, M. D., Houston, Texas

SARCOMAS of the esophagus are rare lesions, comprising about 0.5 per cent of all malignant esophageal neoplasms.[1] Among these mesenchymal tumors, fibrosarcomas occur most commonly, while leiomyosarcomas, arising from smooth muscle elements, are extremely rare.

The earliest reported case of leiomyosarcoma of the esophagus was that of Howard[6] in 1902. The patient, a 51 year old man, complained of difficulty in swallowing of several weeks' duration. He died of inanition soon after admission to the hospital, and at necropsy a diffuse, infiltrating lesion was found in the distal one-third of the esophagus. The mucosa in this area was ulcerated, and there were metastases to the regional lymph nodes and stomach. The neoplasm infiltrated all of the muscle coats, but "at the seat of the tumor" only smooth muscle tissue was found. The microscopic pattern was compatible with leiomyosarcoma.

Von Hacker[4] in 1908 reported Eppinger's case of leiomyosarcoma of the esophagus. The tumor was polypoid and filled the lower end of a dilated esophagus. There were no metastases.

The third reported case was presented by Mallory[9] from the records of the Massachusetts General Hospital. The patient's symptoms were of ten years' duration and were ascribed to a large hiatal hernia. At necropsy the neoplasm arose from the distal esophagus and extended away from, rather than into, the lumen. There were metastases to the regional lymph nodes.

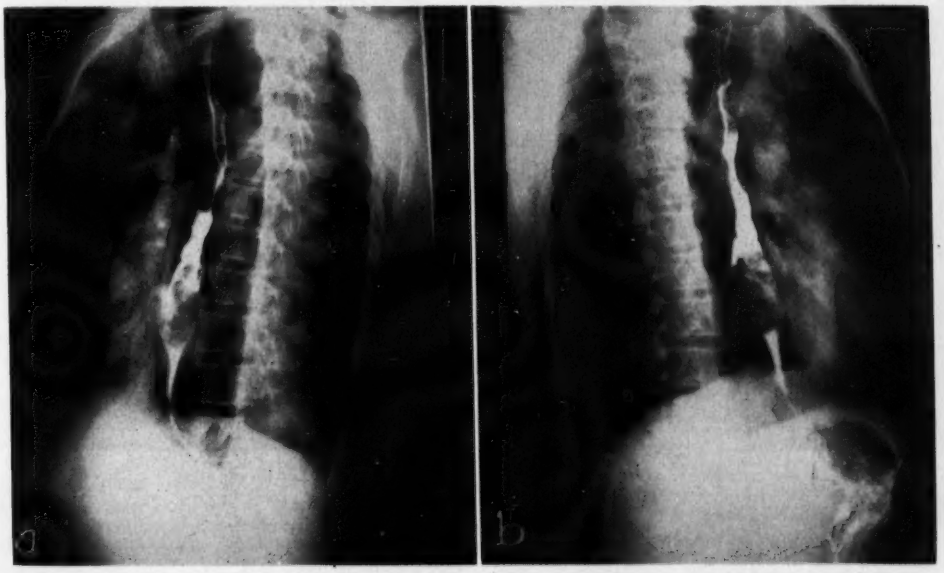

FIG. 1. Left *(a)* and right *(b)* anterior oblique chest roentgenograms with barium swallow suggest a large polypoid neoplasm of the lower esophagus.

Menne and Birge[10] reported the appearance of a leiomyosarcoma in the upper third of the esophagus. Symptoms were of seven years' duration. The tumor produced a mass in the neck beginning at the larynx that extended beneath the sternum. Radiotherapy was given but did not alter the course of the disease, and the patient died. At necropsy the sarcoma was papillomatous and arose from the right lateral wall of the esophagus.

In the case reported by French and Garland,[2] diag-

*From the Departments of Surgery and Pathology, Baylor University College of Medicine and the Veterans Administration Hospital.*

nosis of carcinoma of the esophagus had been made from biopsy material removed at esophagoscopy. At necropsy there was a polypoid, pedunculated tumor attached to the posterior wall of the lower esophagus by a narrow pedicle. There was no evidence of metastases. Histologically, the neoplasm was a sarcoma arising from smooth muscle.

In the case described by Pennes,[14] dysphagia had been present intermittently for about six months. On chest roentgenogram there was a mass in the right side of the posterior mediastinum and the esophagus was displaced to the left. Following a barium swallow, the roentgenogram disclosed several filling defects in the esophagus. A biopsy specimen of the lesion was obtained through the esophagoscope, and microscopically the lesion was a smooth muscle sarcoma. The patient was considered inoperable and was treated with radiotherapy.

There were 2 cases of leiomyosarcoma in a report by Harrington[5] of 6 tumors of the esophagus treated surgically. Both lesions were polypoid and situated in the lower third of the esophagus. One of the patients was treated successfully by esophageal resection and esophagogastrostomy. This is apparently the first recorded instance of surgical resection of this type of neoplasm.

Subsequently, reports of surgical treatment of leiomyosarcomas were made by Ovens and Russel[12] and by Lyons and Garlock.[8] In the former case a diagnosis of squamous cell carcinoma had been made from esophagoscopic biopsy. However, upon examination of the resected esophagus, there was, in addition to the carcinoma, a polypoid lesion at the lower end of the specimen that microscopically was a leiomyosarcoma. The patient of Lyons and Garlock had experienced dysphagia for one year. On biopsy the lesion had been reported to be a rhabdomyosarcoma. At operation the tumor was soft, sausage-shaped, and attached to the esophagus by a narrow pedicle. Esophagectomy and esophagogastrostomy were performed. Microscopic examination revealed a cellular tumor arising from smooth muscle cells. There were numerous mitoses. The patient was well four years after operation.

Recently, Neil and Horne[11] and Piaggio Blanco and associates[15] each have reported a case of leiomyosarcoma of the esophagus. In both instances the tumors were polypoid, pedunculated lesions arising in the upper and middle thirds respectively of the esophagus.

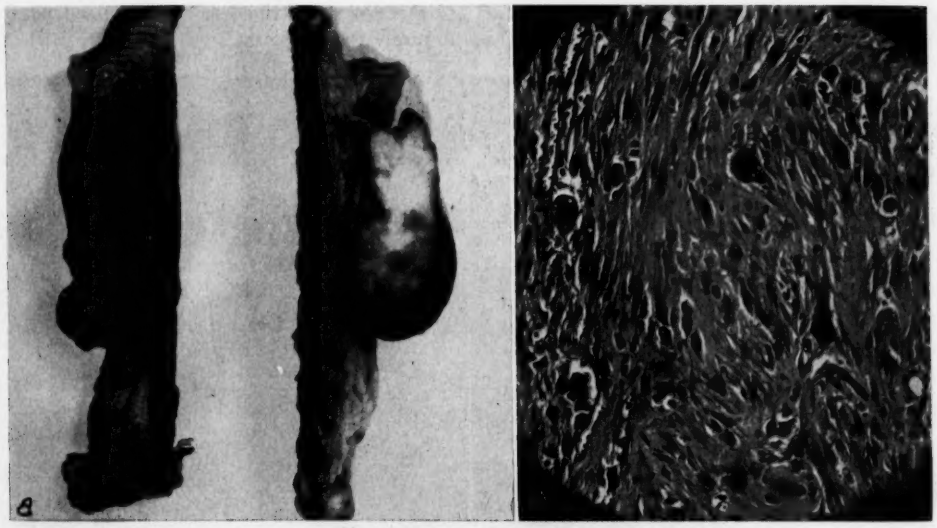

FIG. 2a. Resected esophagus with a large pedunculated tumor projecting from the mucosal surface.

b. Leiomyosarcoma of the esophagus.

We recently have operated upon a patient with an obstructing lesion of the esophagus that proved to be a leiomyosarcoma. Because of the apparent rarity of this type of neoplasm, the case is reported in detail.

## CASE REPORT

A 62 year old Negro man was admitted to the Houston Veterans Administration Hospital on June 11, 1954, complaining of difficulty in swallowing for about three months. The dysphagia consisted principally of a burning substernal and epigastric pain precipitated particularly by the ingestion of solid food and was relieved somewhat by the ingestion of alkalies and upon the patient's assuming a supine position. The patient's oral intake had been limited to liquids for two months prior to admission and during this time he

had lost about 18 pounds. On several occasions he had vomited shortly after ingestion of food.

Physical examination revealed a well-developed, poorly nourished Negro man with evidence of recent weight loss. The blood pressure was 130/90, pulse 72, and temperature 98.8 F. Examination of the respiratory and cardiovascular systems revealed no significant abnormalities. There was slight tenderness in the epigastrium, but no masses were palpable. Laboratory findings were as follows: erythrocytes 4.1 million, hemoglobin 12.8 Gm., hematocrit 41 per cent, leukocytes 12,200 with a normal differential, prothrombic activity 85 per cent. A bromsulphalein excretion test revealed 11 per cent retention of dye in thirty minutes. Repeated stool examinations for occult blood were negative.

On admission, the postero-anterior chest roentgenogram was within normal limits. A barium study of the esophagus on June 14 revealed a large tumefaction in the lower one-half of the esophagus that produced no obstruction to the passage of barium. There was widening of the portion of the esophagus occupied by the filling defect, but there was no proximal dilatation. The roentgenographic appearance was that of a large polypoid neoplasm with superficial erosion or ulcerations in the esophagus adjacent to it (fig. 1). Examination of the remainder of the gastrointestinal tract revealed no abnormalities. The roentgenographic diagnosis was carcinoma of the esophagus, although a benign esophageal neoplasm was suggested because of the intact mucosal pattern.

On June 25, esophagoscopy revealed a granular, friable tumor, the upper extent of which was about 34 cm. from the upper incisor teeth. The neoplasm was movable within the lumen of the esophagus and appeared to be attached by a narrow pedicle. A specimen removed for biopsy was interpreted as leiomyosarcoma.

On June 28, operation was performed. The esophagus was approached through an incision in the right fourth intercostal space. The lung was retracted anteriorly, the mediastinal pleura incised behind the lung root, and the esophagus exposed. A freely movable, sausage-shaped mass could be palpated within the lumen of the middle third of the esophagus. Several anthracotic mediastinal nodes were adjacent to the involved portion of the esophagus, but grossly these did not appear to be malignant. Since the lesion appeared resectable, the esophagus was mobilized from the diaphragmatic hiatus to the azygos vein. A midline abdominal incision was made from the xyphoid process to the umbilicus, and the gastrohepatic and gastrocolic ligaments divided. Care was taken to preserve the right gastric and right gastroepiploic arteries. The left gastric artery was ligated and divided near its origin, thus completely mobilizing the stomach. Pyloromyotomy was performed, and the stomach was drawn into the thorax through the esophageal hiatus. The esophagus was resected proximally from a point just above the azygos vein and distally to include the cardiac end of the stomach. The cardia of the stomach was closed and an end-to-side esophagogastrostomy performed. Upon completion of the anastomosis, the wall of the stomach was sutured to the parietal pleura with several interrupted sutures. The lung was expanded, and the chest and abdominal wounds were closed in layers.

*Pathology.*—The specimen consisted of a portion of esophagus 16 cm. long, opened along its entire length. In the center was a freely movable, pedunculated, lobulated mass 5 by 3.5 by 2.4 cm., attached by a soft pedicle (fig. 2a). The cut surfaces were gray-white and firm. The neoplasm did not extend into the muscular coat of the esophagus.

On microscopic examination the surface was partly covered with stratified squamous epithelium that became narrowed and eventually disappeared over the neoplastic tissue. The neoplastic cells were spindle shaped and in a whorl-like arrangement (fig. 2b). They varied in size, many being up to 50 microns in diameter. The nuclei were large, frequently bizarre, with many in a state of division. Small areas of hemorrhage and necrosis were noted. Trichrome stains verified the smooth muscle origin of the neoplastic cells.

The patient was maintained on nasogastric suction for seventy-two hours, after which he began to take fluids

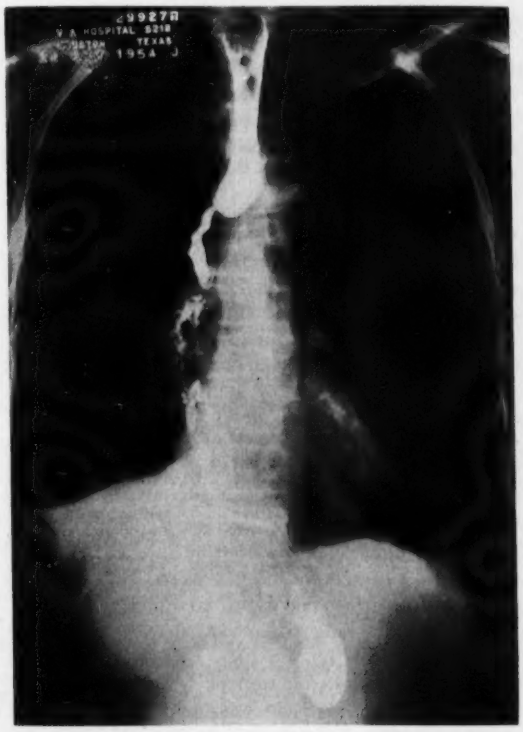


FIG. 3. A chest roentgenogram with barium swallow made two months postoperative.

orally. On the thirteenth postoperative day he began to regurgitate ingested food. Esophagoscopy revealed a large meat bolus completely obstructing the esophagogastric stoma. This was removed with complete relief of symptoms, and he was discharged on July 12. About two months later he returned to the hospital complaining of moderate difficulty in swallowing, and on esophagoscopy a narrowing of the esophagogastric junction was noted. This was readily dilated with a number 32 bougie, with relief of symptoms. His subsequent course has been satisfactory (fig. 3).

## COMMENT

The small number of reported cases of leiomyosarcoma of the esophagus does not permit statistical analysis; however, it is possible to reconstruct from this material some of the clinical and pathologic fea-

tures of the condition (table 1). For 12 cases in which the age of the patient was known, the average age was 57 years, with extremes of 38 and 70 years. Thus, leiomyosarcoma seems to appear at a slightly earlier age than carcinoma of the esophagus.[7, 13]

Grossly two types of leiomyosarcomas of the esophagus have been encountered, namely, polypoid and infiltrating. Polypoid tumors occurred in 9 cases, whereas 4 cases were infiltrating. Usually the polypoid neoplasms were attached to the esophageal wall by a narrow pedicle that permitted movement of the tumor. More than half of the tumors were situated in the lower esophagus in contrast to squamous cell carcinoma which occurs primarily in the midesophagus.[3, 7, 13, 16]

In only 3 of 10 cases of leiomyosarcoma had the neoplasm extended beyond the esophagus, whereas in carcinoma of the esophagus, particularly of the upper and middle thirds, there almost invariably is extension. In 2 cases the tumor had spread to the mediastinal lymph nodes, and in the third case there was local extension through the wall of the esophagus into the adjacent mediastinal tissue.

Symptomatically, leiomyosarcoma is probably indistinguishable from carcinoma since both neoplasms cause difficulty in swallowing. There is, however, a striking difference in the duration of symptoms. In 12 cases of leiomyosarcoma the shortest duration of symptoms was one month and the longest ten years with an average of two years. With esophageal carcinoma the average duration of symptoms is about four months.[7] In view of the longer duration of symptoms and the apparent tendency to remain confined to the esophagus for long periods of time, leiomyosarcoma of the esophagus is probably a slowly growing neoplasm.

The roentgenographic features of leiomyosarcoma are fairly characteristic and consist of an obstructing polypoid lesion with an intact mucosal surface. This latter characteristic distinguishes the lesion from carcinoma of the esophagus, which usually is ulcerated.

## SUMMARY

Twelve cases of leiomyosarcoma of the esophagus have been collected from the literature and 1 additional case is added. Leiomyosarcoma is a slowly growing neoplasm occurring in a somewhat younger age group than carcinoma and producing symptoms for prolonged periods. The lesion is generally polypoid and attached to the wall of the esophagus by a narrow pedicle, and extension beyond the esophagus is uncommon. These features make this lesion a suitable one for surgical resection. Three instances of successful removal of leiomyosarcomas have been reported previously, and to these is added the case reported here.

TABLE 1.—*Data on 13 Cases of Leiomyosarcoma of the Esophagus.*

| Author | Year Reported | Patient: Age | Patient: Sex | Duration Symptoms | Neoplasm: Type | Neoplasm: Location | Neoplasm: Metastasis |
|---|---|---|---|---|---|---|---|
| Howard | 1902 | 51 | M | Several wk. | Infiltrating | Distal third | . . . . . |
| von Hacker | 1908 | .. | .. | .... | Polypoid | Distal third | None |
| Mallory | 1935 | 58 | M | 10 yr. | Infiltrating | Distal third | Regional lymph nodes |
| Menne & Birge | 1937 | 38 | M | 7 yr. | Infiltrating | Upper third | . . . . . |
| French & Garland | 1941 | 70 | M | 2 mo. | Polypoid | Distal third | None |
| Pennes | 1942 | 53 | M | 6 mo. | Infiltrating | Middle third | . . . . . |
| Harrington | 1949 | 60 | F | 1 yr. | Polypoid | Distal third | None |
| Harrington | 1949 | 60 | F | 3 wk. | Polypoid | Distal third | Local extension |
| Ovens & Russel | 1951 | 56 | F | 8 mo. | Polypoid | Distal third | None |
| Lyons & Garlock | 1951 | 58 | F | 1 yr. | Polypoid | Lower two-thirds | None |
| Blanco et al. | 1952 | 65 | F | 1 yr. | Polypoid | Middle third | Mediastinal |
| Neil & Horne | 1953 | 54 | M | 1 mo. | Polypoid | Upper third | None |
| Creech, Overton, & Erickson | 1955 | 62 | M | 3 mo. | Polypoid | Middle third | None |

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**LEIOMYOSARCOMA—*Creech et al*—*continued***

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# NEWS

## COMING MEETINGS AND CLINICS

Texas Medical Association. Dr. J. Layton Cochran, San Antonio, Pres.; Mr. C. Lincoln Williston, 1801 North Lamar Blvd., Austin, Executive Secy.

American Medical Association, Atlantic City, N. J., June 6-10, 1955. Dr. Walter B. Martin, Norfolk, Va., Pres.; Dr. George F. Lull, 535 North Dearborn St., Chicago 10, Secy.

NATIONAL AND REGIONAL

American Academy of Allergy. Dr. Stanley F. Hampton, St. Louis, Pres.; Dr. Frances C. Lowell, 65 E. Newton St., Boston, Secy.

American Academy of Dermatology and Syphilology, Chicago, Dec. 3-8, 1955. Dr. Arthur C. Curtis, Ann Arbor, Pres.; Dr. James R. Webster, 55 E. Washington St., Chicago 2, Secy.

American Academy of General Practice. Dr. W. B. Hildebrand, Menasha, Wis., Pres.; Mr. Mac F. Cahal, 406 W. 34th St., Kansas City 2, Executive Secy.

American Academy of Obstetrics and Gynecology, Chicago, Dec. 13, 1955. Dr. William F. Mengert, Dallas, Pres.; Dr. C. Paul Hodgkinson, 116 S. Michigan Blvd., Chicago 3, Secy.

American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 9-14, 1955. Dr. Algernon B. Reese, New York, Pres.; Dr. W. L. Benedict, 100 First Ave. Bldg., Rochester, Minn., Secy.

American Academy of Pediatrics, Chicago, Oct. 3-6, 1955. Dr. A. Crawford Bost, San Francisco, Calif., Pres.; Dr. E. H. Christopherson, 610 Church St., Evanston, Ill., Secy.

American Association for Thoracic Surgery. Dr. Edward S. Welles, Saranac Lake, N. Y., Pres.; Dr. Paul C. Samson, 3959 Happy Valley Rd., Lafayette, Calif., Secy.

American Association of Genito-Urinary Surgeons, Monterey, Calif., May 22-25, 1955. Dr. Fletcher H. Colby, Boston, Pres.; Dr. John A. Taylor, 2 E. 45th St., New York 22, Secy.

American Association of Obstetricians, Gynecologists and Abdominal Surgeons, Hot Springs, Va., Sept. 8-10, 1955. Dr. Thaddeus L. Montgomery, Philadelphia, Pres.; Dr. F. R. Lock, Bowman Gray School, Winston-Salem, N. C., Secy.

American Cancer Society. Dr. Guy Aud, Louisville, Ky., Pres.; Mr. M. R. Runyon, 47 Beaver St., New York, Executive Vice-Pres.

American College of Allergists. Dr. Homer Prince, Houston, Pres.; Dr. Fred W. Wittich, 401 La Salle Medical Bldg., Minneapolis 2, Secy.

American College of Chest Physicians, Atlantic City, N. J., June 2-5, 1955. Dr. William A. Hudson, Detroit, Pres.; Mr. Murray Kornfeld, 112 E. Chestnut St., Chicago 11, Executive Secy.

American College of Physicians. Dr. C. C. Sturgis, Ann Arbor, Mich., Pres.; Mr. E. R. Loveland, 4200 Pine St., Philadelphia 4, Secy.

American College of Radiology. Dr. Howard P. Doub, Detroit, Pres.; Mr. W. C. Stronach, 20 N. Wacker Drive, Chicago 6, Executive Secy.

American College of Surgeons, Chicago, Oct. 31-Nov. 4, 1955. Dr. Alfred Blalock, Baltimore, Pres.; Dr. Michael L. Mason, 40 E. Erie St., Chicago 11, Secy.

American Congress on Obstetrics and Gynecology. Dr. R. Gordon Douglas, 116 S. Michigan Ave., Chicago 3, Chairman.

American Congress of Physical Medicine and Rehabilitation, Detroit, Aug. 28-Sept. 2, 1955. Dr. William D. Paul, Iowa City, Pres.; Dr. Frances Baker, 1 Tilton Ave., San Mateo, Calif., Secy.

American Dermatological Association. Dr. Richard S. Weiss, St. Louis, Pres.; Dr. J. Lamar Callaway, Duke Hospital, Durham, N. C., Secy.

American Gastro-Enterological Association, Atlantic City, N. J., June 4-5, 1955. Dr. Dwight L. Wilbur, San Francisco, Pres.; Dr. H. Marvin Pollard, University Hosp., Ann Arbor, Mich., Secy.

American Gynecological Society, Quebec, Canada, May 23-25, 1955. Dr. Philip F. Williams, Philadelphia, Pres.; Dr. John I. Brewer, 104 S. Michigan Ave., Chicago, Secy.

American Heart Association, New Orleans, Oct. 26-30, 1955. Dr. E. Cowles Andrus, Baltimore, Pres.; Mr. Irving Hexter, 44 E. 23rd St., New York 10, Secy.

American Hospital Association, Atlantic City, N. J., Sept. 19-22, 1955. Dr. Frank R. Bradley, St. Louis, Pres.; Dr. Edwin L. Crosby, 18 E. Division St., Chicago, Executive Director.

American Laryngological, Rhinological, and Otological Society. Dr. Kenneth M. Day, Pittsburgh, Pres.; Dr. C. S. Nash, 277 Alexander St., Rochester 7, N. Y., Secy.

American Neurological Association, Chicago, June 13-15, 1955. Dr. Percival Bailey, Chicago, Pres.; Dr. H. Houston Merritt, 710 W. 168th St., New York 32, Secy.

American Ophthalmological Society, W. Sulphur Springs, W. Va., June 2-4, 1955. Dr. Everett L. Goar, Houston, Tex., Pres.; Dr. M. C. Wheeler, 30 W. 59th St., New York 19, Secy.

American Orthopedic Association, W. Sulphur Springs, W. Va., June 19-22, 1955. Dr. J. Warren White, Honolulu, Hawaii, Pres.; Dr. George O. Eaton, 4 E. Madison St., Baltimore 2, Secy.

American Pediatric Society, Quebec, Canada, June 13-17, 1955. Dr. Alfred H. Washburn, Denver, Pres.; Dr. A. C. McGuinness, 237 Medical Laboratory, University of Pennsylvania, Philadelphia 46, Secy.

American Proctologic Society, New York, June 1-4, 1955. Dr. A. W. Martin Marino, Brooklyn, N. Y., Pres.; Dr. Karl Zimmerman, 3500 Fifth Ave., Pittsburgh 13, Secy.

American Psychiatric Association, Atlantic City, N. J., May 9-13, 1955. Dr. Alfred P. Noyes, Norristown, Pa., Pres.; Dr. William Malamud, 80 E. Concord St., Boston 18, Secy.

American Public Health Association, Kansas City, Mo., Nov. 14-18, 1955. Dr. Herman E. Hilleboe, Albany, N. Y., Pres.; Dr. R. M. Atwater, 1790 Broadway, New York 19, Executive Secy.

American Society of Anesthesiologists, Boston, Oct. 29-Nov. 3, 1955. Dr. B. B. Sankey, Cleveland, Ohio, Pres.; Dr. J. E. Remlinger, Jr., 188 W. Randolph St., Chicago, Secy.

American Society of Clinical Pathologists. Dr. John R. Schenken, Omaha, Pres.; Dr. Clyde G. Culbertson, 1040 W. Michigan St., Indianapolis 6, Secy.

American Surgical Association. Dr. John H. Gibbon, Jr., Philadelphia, Pres.; Dr. R. Kennedy Gilchrist, 59 East Madison, Chicago 3, Secy.

American Urological Association, Los Angeles, May 16-19, 1955. Dr. W. Joseph McMartin, Omaha, Neb., Pres.; Dr. C. H. deT. Shivers, 121 S. Illinois Ave., Atlantic City, N. J., Secy.

Association of American Physicians and Surgeons. Dr. Thomas G. Goldsmith, Greenville, S. C., Pres.; Mr. Harry E. Northam, 185 N. Wabash Ave., Chicago 1, Executive Secy.

International College of Surgeons, U. S. Chapter, Chicago, Sept. 7-10, 1955. Dr. William R. Lovelace, Albuquerque, N. M., Pres.; Dr. Karl Meyer, 1516 Lake Shore Drive, Chicago, Secy.

National Tuberculosis Association, Milwaukee, May 23-27, 1955. Dr. John H. Skavlem, Cincinnati, Pres.; Mrs. Morrell DeReign, 1790 Broadway, New York 19, Secy.

Radiological Society of North America, Chicago, Dec. 11-16, 1955. Dr. Tom B. Bond, Fort Worth, Pres.; Dr. D. S. Childs, 713 E. Genesee, Syracuse 2, N. Y., Secy.

Southern Medical Association, Houston, Texas, Nov. 14-17, 1955. Dr. Robert L. Sanders, Memphis, Tenn., Pres.; Mr. V. O. Foster, 1020 Empire Bldg., Birmingham 3, Ala., Secy.

Southern Psychiatric Association. Dr. John D. Trawick, Louisville, Ky., Pres.; Dr. Joseph L. Knapp, 210 N. Westmoreland, Dallas, Secy.

Southern Surgical Association, Hot Springs, Va., Dec. 6-8, 1955. Dr. Carrington Williams, Richmond, Va., Pres.; Dr. George Finney, 2947 St. Paul St., Baltimore, Secy.

Southwest Allergy Forum. Dr. Henry D. Ogden, New Orleans, Pres.; Dr. Stanley Cohen, 1441 Delachaise St., New Orleans, Secy.

Southwest Regional Cancer Conference, Fort Worth. Dr. John L. Wallace, Box 1719, Fort Worth, Chm.

Southwestern Medical Association, Phoenix, Ariz., November, 1955. Dr. Joseph Bank, Phoenix, Pres.; Dr. Celso C. Stapp, 800 Montana, El Paso, Secy.

Southwestern Surgical Congress, Kansas City, Mo., Sept. 12-14, 1955. Dr. Lawrence P. Engel, Kansas City, Mo., Pres.; Dr. C. M. O'Leary, 207 Plaza Court Bldg., Oklahoma City, Secy.

Tri-State Medical Society, Texarkana, September, 1955. Dr. William B. Harrell, Texarkana, Pres.; Dr. Karlton Kemp, 408 Hazel, Texarkana, Ark., Secy.

United States-Mexico Border Public Health Association, Mexico, D. F., May 6-9, 1955. Mr. Richard F. Poston, San Francisco, Pres.; Dr. Sidney B. Clark, 314 U. S. Court House, El Paso, Secy.

STATE

Private Clinics and Hospitals Association of Texas, Austin. Dr. Neil Buie, Marlin, Pres.; Mr. C. H. Rugeley, Wharton, Secy.

Texas Academy of General Practice, Fort Worth, Sept. 18-21, 1955. Dr. L. Bonham Jones, San Antonio, Pres.; Dr. Woodson W. Harris, 308 W. 15th St., Austin, Secy.

Texas Academy of Internal Medicine. Dr. Martin S. Buehler, Dallas, Pres.; Dr. George M. Jones, 1314 Medical Arts Bldg., Dallas, Secy. Meetings restricted to members.

Texas Air-Medics Association. Dr. W. A. Ostendorf, Fort Worth, Pres.; Dr. C. F. Miller, P. O. Box 1338, Waco, Secy.

Texas Association of Blood Banks, Houston, Dec. 8-10, 1955. Dr. Jarrett E. Williams, Abilene, Pres.; Miss Marjorie Saunders, 3500 Gaston Ave., Dallas, Secy.

Texas Association of Obstetricians and Gynecologists. Dr. John Delany, Galveston, Pres.; Dr. Oran V. Prejean, 4317 Oak Lawn, Dallas, Secy.

Texas Chapter, American College of Chest Physicians, Galveston, 1956. Dr. Samuel Topperman, Tyler, Pres.; Dr. J. O. Armstrong, 3810 Swiss Ave., Dallas, Secy.

Texas Club of Internists. Dr. Victor E. Schulze, San Angelo, Pres.; Dr. Charles Darnall, Capital National Bank Bldg., Austin, Secy.

Texas Dermatological Society, Houston, Nov. 14-17, 1956. Dr. E. B. Richie, Galveston, Pres.; Dr. Thomas L. Shields, 1216 Pennsylvania Ave., Fort Worth, Secy.

Texas Diabetes Association. Dr. Mavis P. Kelsey, Houston, Pres.; Dr. Hugo Engelhardt, P. O. Box 2180, Houston, Secy.

Texas Division, American Cancer Society. Mr. Travis Wallace, Dallas, Pres.; Mr. Curt W. Reimann, 1609 Colorado, Austin, Acting Executive Director.

Texas Geriatric Society. Dr. Henry H. Niehuss, Longview, Pres.; Dr. Frank V. Mondrik, 214 Bramlette Bldg., Longview, Secy.

Texas Heart Association. Dr. George R. Herrmann, Galveston, Pres.; Mr. Edgar M. Brown, 404 Jesse H. Jones Library Bldg., Texas Medical Center, Houston 25, Executive Director.

Texas Hospital Association, Dallas, April 3-5, 1956. Mr. Boone Powell, Dallas, Pres.; Mrs. Ruth Barnhart, 2210 Main St., Dallas, Secy.

Texas Neuropsychiatric Association. Dr. Stephen Weisz, Dallas, Pres.; Dr. Bruce H. Beard, 1519 Pennsylvania, Fort Worth, Secy.

Texas Orthopedic Association. Dr. Paul Williams, Dallas, Pres.; Dr. Margaret Watkins, 3629 Fairmount St., Dallas, Secy.

Texas Pediatric Society, Galveston, October 21-22, 1955. Dr. M. C. Carlisle, Waco, Pres.; Dr. James N. Walker, 3616 Tulsa Way, Fort Worth, Secy.

Texas Proctologic Society, Galveston, February, 1956. Dr. John McGivney, Galveston, Pres. and Secy.

Texas Public Health Association, Fort Worth, Feb. 26-29, 1956. Mr. Ed Riedel, Austin, Pres.; Mr. H. E. Drumwright, City Health Department, Dallas, Executive Secy.

Texas Radiological Society, Fort Worth, Jan. 20-21, 1956. Dr. Martin Schneider, Galveston, Pres.; Dr. R. P. O'Bannon, 650 Fifth Ave., Fort Worth, Secy.

Texas Railway and Traumatic Surgical Association. Dr. Raleigh White, Temple, Pres.; Dr. W. D. Marrs, 306 Broadway, Fort Worth, Secy.

Texas Rheumatism Association, San Antonio, Dec. 9, 1955. Dr. Charles H. Cornwell, Marlin, Pres.; Dr. Warren W. Moorman, 901 W. Leuda, Fort Worth, Secy.

Texas Society for Mental Health, San Antonio, 1956. Dr. Abe Hauser, Houston, Pres.; Mr. John Lane, 2510 San Antonio, Austin, Acting Executive Secy.

Texas Society of Anesthesiologists. Dr. Frank O. Barrett, El Paso, Pres.; Dr. Milton M. Rosenzweig, 200 Wildwood Dr. E., San Antonio, Secy.

Texas Society of Gastroenterologists and Proctologists. Dr. Charles Hardwicke, Austin, Pres.; Dr. W. T. Arnold, 1402 Hermann Prof. Bldg., Houston, Secy.

Texas Society of Ophthalmology and Otolaryngology. Dr. John L. Matthews, San Antonio, Pres.; Dr. Gatlin Mitchell, 1604 Medical Arts Bldg., Fort Worth, Secy.

Texas Society of Pathologists. Dr. C. B. Sanders, Houston, Pres.; Dr. M. H. Grossman, St. Paul Hospital, Dallas, Secy.

Texas Surgical Society. Dr. Dudley Jackson, Sr., San Antonio, Pres.; Dr. Albert W. Hartman, 414 Navarro St., San Antonio 5, Secy.

Texas Tuberculosis Association, Midland, April 6-7, 1956. Mrs. Joella Terrill Butler, Wichita Falls, Pres.; Miss Pansy Nichols, 2406 Manor Rd., Austin, Executive Secy.

Texas Urological Society, Austin, February, 1956. Dr. A. J. Ashmore, Corpus Christi, Pres.; Dr. Rex Carter, Austin, Secy.

DISTRICT

First District Society. Dr. Delphin von Briesen, El Paso, Pres.; Dr. W. G. Morrow, Jr., First National Bldg., El Paso, Secy.

Second District Society, Odessa, April 19, 1956. Dr. T. W. Novak, Odessa, Pres.; Dr. Willis T. Carson, 506 North Allegheny, Odessa, Secy.

Third District Society. Dr. M. C. Overton, Jr., Pampa, Pres.; Dr. William Klingensmith, 215 Fisk Bldg., Amarillo, Secy.

Fourth District Society, Brownwood, Oct. 20, 1955. Dr. James N. White, San Angelo, Pres.; Dr. Joe B. Stephens, Bangs, Secy.

Fifth and Sixth Districts Society, Corpus Christi, July 7-9, 1955. Dr. John J. Sloan, Corpus Christi, Pres.; Dr. E. Jackson Giles, Medical Center, Suite 42, Corpus Christi, Secy.

Seventh District Society. Dr. William McLean, Austin, Pres.; Dr. John Rainey, 1709 San Antonio, Austin, Secy.

Eighth District Society, Galveston, 1955. Dr. George E. Glover, Jr., Victoria, Pres.; Dr. York Lancaster, Port Lavaca, Secy.

Ninth District Society, Baytown. Dr. Joseph T. Dabney, Livingston, Pres.; Dr. Lyman C. Blair, 1212 Rothwell, Houston, Secy.

Tenth District Medical Society, Lufkin, May 25. Dr. J. C. Klein, Lufkin, Pres.; Dr. Rider Stockdale, Jasper, Secy.

Eleventh District Society. Dr. Porter Bailes, Tyler, Pres.; Dr. Hugh F. Rives, Jacksonville, Secy.

Twelfth District Society, Temple, July, 1955. Dr. R. H. Harrison, Jr., Bryan, Pres.; Dr. J. H. Johnson, 304 South 22nd, Temple, Secy.

Thirteenth District Society. Dr. P. M. Kuykendall, Ranger, Pres.; Dr. Robert D. Moreton, 815 Medical Arts Bldg., Fort Worth, Secy.

Fourteenth District Society. Dr. J. David Thomas, Denton, Pres.; Dr. L. W. Johnston, 502 W. College St., Terrell, Secy.

Fifteenth District Society, Marshall, 1956. Dr. James Harris, Marshall, Pres.; Dr. L. E. Rutledge, Daingerfield, Secy.

CLINICS

Dallas Southern Clinical Society. Dr. Lawrence B. Sheldon, Dallas, Pres.; Miss Helga Boyd, Medical Arts Bldg., Dallas 1, Executive Secy.

Central Texas Spring Clinic, Waco, 1956. Dr. James T. Archer, Meridian, Pres.; Dr. Milton Spark, 121 Dallas St., Waco, Secy.

International Medical Assembly of Southwest Texas, San Antonio. Dr. John M. Smith, Jr., 205 Camden St., San Antonio, Secy.

New Orleans Graduate Medical Assembly, New Orleans, Feb. 27-March 1, 1956. Dr. Maurice E. St. Martin, Room 103, 1430 Tulane Ave., New Orleans 12, Secy.

North Texas-Southern Oklahoma Fall Clinical Conference. Dr. L. N. Simmons, 1518 Tenth St., Wichita Falls, Chairman.

Oklahoma City Clinical Society Conference, Oklahoma City, Oct. 24-27, 1955. Miss Alma F. O'Donnell, 512 Medical Arts Bldg., Oklahoma City 2, Executive Secy.

Postgraduate Medical Assembly of South Texas, Houston, July 18-20, 1955. Dr. C. Forrest Jorns, 5644 Lawndale, Houston, Secy.

State Tumor Conference. Dr. Bailey R. Collins, 925½ Scott Street, Wichita Falls, Director.

BOARD EXAMINATIONS

Texas State Board of Examiners in Basic Sciences. Mrs. Betty Ratcliff, 407 Perry-Brooks Bldg., Austin, Chief Clerk.

Texas State Board of Medical Examiners, Fort Worth, June 20-22, 1955. Dr. M. H. Crabb, 1714 Medical Arts Bldg., Fort Worth, Secy.

## PERSONALS

*Dr. J. G. Rodarte,* Temple, presented a paper at the seventh annual hospital pharmacy seminar conducted by the University of Texas College of Pharmacy.

Three faculty members of the Baylor University College of Medicine, *Drs. George L. Jordan,* assistant professor of surgery; *Joseph A. Stool,* assistant professor of pediatrics; and *Harold L. Dobson,* instructor in medicine, were guest lecturers at a one-day postgraduate study course sponsored by the Texas Academy of General Practice and the three Texas medical schools. The course, designed for family doctors, was held in Beaumont.

*Dr. William F. Mengert,* Dallas, has received the seventh annual Marchman medical award for outstanding scientific contribution and distinguished service to medicine. The award was presented by the Dallas Southern Clinical Society and is named in honor of Dr. Oscar M. Marchman, Sr., first president of the society.

A fellowship to the M. D. Anderson Hospital and Tumor Institute for the study of malignant disease has been established in honor of *Dr. Dudley Jackson,* San Antonio.

Five Texas doctors have been certified in psychiatry by the American Board of Psychiatry and Neurology at the latest examination. They are *Dr. George Andrew Constant,* Victoria; *Dr. James Edward Hamill,* Port Arthur; *Dr. Floy Jack Moore,* Houston; *Dr. William Flavel Sheeley,* Randolph Field; and *Dr. Warren Scott Williams,* Galveston.

*Dr. Bernard T. Fein,* San Antonio, has been elected to fellowship in the American Academy of Allergy.

*Dr. Clinton M. Shaw, Jr.*, Wichita Falls, has been certified by the American Board of Internal Medicine.

*Dr. James D. Murphy*, Fort Worth, has been elected speaker of the Congress of Delegates of the American Academy of General Practice at the recent convention in Los Angeles.

*Drs. Albert M. Goggans, John F. Lindsay, Frank J. Blaha,* and *E. B. Wilson, Jr.*, all of Fort Worth, were panel members at a public discussion of high blood pressure. The meeting was sponsored by the Tarrant County Medical Society.

*Dr. Paul Bontley* is the new president of the Arlington Medical and Dental Society.

*Dr. Vincent Cox*, Marlin Veterans Hospital, has been named president of the Marlin Rotary Club.

*Dr. Stanley Clayton* is the new vice-president of the Dad's Club of the Houston School in Denison.

*Dr. H. F. Connally, Jr.*, has served as mayor of Waco for the past year, and stepped down from his office April 19.

*Dr. Wayne Baden*, Raymondville, has been appointed one of the regional chairmen of the United States Branch of the International Fertility Association.

*Dr. James D. Gossett*, Rankin, is the new president of the Upton County Hospitals Staff.

*Dr. Harry M. Shytles, Jr.*, medical consultant at Bonham Veterans Hospital, has been named Sherman's Outstanding Young Man of 1954.

*Dr. Shell H. Townsend* is the new president of the Childress City Planning Council.

*Dr. James E. Reed*, Madisonville, is serving as 1955 president of the local Rotary Club.

*Dr. Jack Wilson Flowers*, Waco, was married to Miss Helen Ferguson, Marlin, on March 18 in Marlin. *Dr. Thomas Elton Linstrum* and Miss Margie J. Holcombe were married March 5 in Kerrville. *Dr. Hal W. Maxwell* was married to Miss Coral Lindsey on January 29 in Dallas.

A boy, Michael Emmett, was born to *Dr. and Mrs. Emmett N. Wilson, Jr.*, Pearsall, on February 21.

*Dr. and Mrs. S. V. Yeakel*, Amarillo, are the parents of a girl born early in March.

## POSTGRADUATE COURSES OFFERED

*Radioisotopic Techniques*—University of Texas Postgraduate School of Medicine, May 5-7. Part I consisted of orientation in radioisotopic techniques for medical technologists. Radiologic safety measures and the principles and employment of instruments used were emphasized. Registration was limited to twenty. Part II provides actual laboratory practice and experience in selected techniques. The time of Part II, its location, and the duration of the training is flexible, depending on the particular techniques in which the technologist desires to acquire facility.

*Dermatology and Endocrinology*—University of Texas Postgraduate School of Medicine, El Paso Division, April 17. The course covered dermatological allergy, common dermatoses, disseminated arteritis, hyperthyroidism, and endocrinological aspects of the posterior pituitary and hypothalamic disease.

*Ivan H. Mattson I Memorial Tuberculosis Seminar*—Southwestern Medical School, Dallas, June 13-14. The course is designed for general practitioners, and its scope the tuberculin test, laboratory findings, and roentgen-rays to the question of home care or sanatorium rest, after-care, and question of home care or sanatorium rest, after-care, and rehabilitation. Problems of public health responsibility and the social and psychiatric aspects will also be discussed. Climaxing the course will be an roentgen-ray conference on problems submitted by registrants.

*Laboratory Refresher Training Courses*—Laboratory Branch of the Communicable Diseases Center, Chamblee, Ga., July, 1955, through June, 1956. Nineteen courses in laboratory techniques and diagnosis will be offered during the period. Further information may be obtained from the Laboratory Training Services, Communicable Disease Center, U. S. Public Health Service, P. O. Box 185, Chamblee, Ga.

## TEXAS PUBLIC HEALTH ASSOCIATION

The Texas Public Health Association met in Galveston on February 15. The relationship between animal health and public health was discussed by James H. Steele, director of the Veterinary Public Health Section of the communicable diseases section, Atlanta. Other speakers on the program were Dr. Dean F. Winn, director of the hospital survey and construction division of the Texas State Department of Health; Dr. J. W. Bass, director of the Dallas city health department; Dr. F. J. L. Blasingame, Wharton, President of the Texas Medical Association; Dr. J. B. Copeland, San Antonio, chairman of the State Board of Health; and Dr. Mayhew Derryberry, Washington, D. C., chief of the public health education branch of the United States Public Health Service.

### AWARDS FOR GRADUATE TRAINING

Mead Johnson awards for graduate training are now available. The awards vary from one specialty to another; in most instances the awards are for $1,000 each. Information about awards within the various specialty fields may be obtained by writing the executive secretaries of the American Academy of General Practice, the American College of Physicians, the American Academy of Obstetrics and Gynecology, the American Academy of Pediatrics, or the American College of Surgeons.

### AMERICAN PROCTOLOGIC SOCIETY

General Principles of Water and Electrolyte Balance in Gastrointestinal Tract Surgery—Dr. Henry T. Randall, New York City.

Autonomic Nervous System of Colon and Rectum—Dr. Benjamin G. Shafiroff, Brooklyn.

Pharmacology of Local Anesthetic Agents—Dr. E. A. Rovenstine, New York City.

Rationale for Prophylactic and Therapeutic Employment of Antibiotics as Adjunct to Surgery of Alimentary Tract—Dr. Frank L. Meleny, New York City.

The above program will be presented at the annual meeting of the American Proctologic Society to be held in New York City, June 1-4, 1955.

### SCHERING AWARD COMPETITION

The tenth annual Schering Award competition for medical students has opened. The deadline for entry forms specifying the choice of title is July 1, and manuscripts must be mailed not later than October 1. Students may compete individually or in teams. Titles of three subjects on which students are invited to submit papers are: "Current Concepts in the Management of Osteoporosis," "Prevention and Treatment of Blood Transfusion Reactions," and "Recent Trends in the Clinical Use of Adrenocortical Steroids." Further information may be obtained from the Schering Award Committee, 60 Orange Street, Bloomfield, N. J.

### Texas Club of Internists

The Texas Club of Internists met February 25 and 26 in Houston. New officers elected are Dr. Charles Barrier, Fort Worth, president; Dr. Hatch Cummings, Houston, vice-president; and Dr. Charles Darnall, Austin, secretary. The fall meeting will be held at the Mayo Clinic.

### TEXAS GIRL PLACES IN NATIONAL ESSAY CONTEST

Miss Betty Wood, Wichita Falls, won second prize, $500, in the ninth annual national essay contest sponsored by the Woman's Auxiliary. Miss Wood's essay on "The Advantages of Private Medical Care" won first place in the state contest. Miss Wood was present at the annual session in Fort Worth, and was congratulated by Dr. F. J. L. Blasingame, President of the Texas Medical Association.

## Treatment of Mass Casualties Studied

The Brooke Army Medical Center was host to sixteen educators, members of the Medical Education for National Defense committee, who came to study Army medical procedures in order to incorporate certain military instruction in medical school courses. Care and treatment of mass casualties was emphasized, and the visitors observed the medical aspects of an infantry division in an attack. Surgical methods used in forward combat areas and the latest Army burn treatment techniques were discussed. Committee members making the visit were from twelve leading medical schools in the United States.

### UNIVERSITY OF TEXAS MEDICAL BRANCH

*Dr. William J. Kerr,* emeritus professor of medicine at the University of California Medical Center, San Francisco, gave a special talk to the staff and students of the University of Texas Medical Branch on April 30 on "The Hyperventilation Syndrome."

*The annual lecture sponsored by Alpha Omega Alpha,* honorary medical society, was given at the Medical Branch recently by Dr. Walter B. Martin, Norfolk, President of the American Medical Association.

*Dr. F. J. L. Blasingame,* Wharton, Past President of the Texas Medical Association, will be commencement speaker for more than 150 graduates of the University of Texas Medical Branch in ceremonies June 3 in Galveston. Dr. Blasingame is president of the local chapter of Alpha Omega Alpha, honorary medical society, and is a lecturer in anatomy at the Medical Branch.

*Included in the April calendar of events* were a pediatrics roentgen-ray conference, a rehabilitation seminar, and an anesthesiology seminar and conference, all of which were held on April 6. Dr. W. D. Forbus, professor of pathology at Duke University, Durham, N. C., spoke the same day on "Tumors as Etiologic Agents."

*The University of Texas Medical Branch* calendar of events lists for April 25 a combined pediatrics, chest surgery, radiology, and cardiac catheterization conference and an anatomy seminar by Dr. J. V. Basmajian, University of Toronto, Canada. His subject was "Electromyographic Studies of Human Spasticity and Chlorpromazine."

### PLASTIC SURGERY CONTEST OPEN

Awards in two classifications are being offered by the Foundation of the American Society of Plastic and Reconstructive Surgery. In the junior classification, a cash prize of $1,000 with a three months' scholarship and a second cash prize of $750 with a three months' scholarship will be given for winning essays on the results of original research in plastic and reconstructive surgery. Residents in training and plastic surgeons who have been in practice no longer than five years are eligible for these awards. In the senior classification, for contestants in practice of the specialty more than five years, a silver plaque or certificate will be awarded a winning essay.

Further information may be obtained from the Awards Committee of the foundation, 30 Central Park South, New York 19, to which manuscripts must be mailed by July 1.

### NEW INSECTICIDE (DDVP) HOLDS PROMISE

A new insecticide, DDVP, is more potent in killing insects and less toxic to humans and farm animals than many modern economic poisons. It was discovered by research scientists at the Savannah, Ga., laboratory of the Public Health Service's Communicable Disease Center. The discovery is in the organic phosphorous insecticide field, and its name is derived from the initials of its chemical name—dimethyl dichloro vinyl phosphate.

DDVP's importance lies in several characteristics which make it different from other insecticides in use today. One of these is its very high potency, although it is much safer for animal and man than other organic phosphorous insecticides. DDVP may prove to be of greatest value where flies and insects have developed a resistance to DDT. Since DDVP is an organic phosphorous insecticide, a different chemical family that was not previously considered as substitutes for DDT and its related compounds, its discovery opens up a whole new class of economic poisons.

In addition to being effective against flies, the discoverers believe that DDVP will prove useful against pests of agricultural crops, especially mites and aphids. Because of its high volatility, DDVP, unlike DDT, will not remain effective over long periods of time. Thus, it will be especially suitable on crops where insecticide residues are objectionable.

### TEXAS HOSPITAL ASSOCIATION

The twenty-sixth annual convention of the Texas Hospital Association was held in Houston April 12-14. Boone Powell, Dallas, assumed the presidency and H. M. Cardwell, Lufkin, was named president-elect. Other officers for 1955 are Bill Burton, El Paso vice-president, and Bolton Boone, Dallas, treasurer. Newly elected trustees are W. P. Earngey, Fort Worth; D. S. Riley, Big Spring; and F. S. Walters, Jr., Amarillo. The 1956 meeting will be held April 3-5 in Dallas.

### TEXAS TUBERCULOSIS ASSOCIATION

Three hundred and fifteen members and guests attended the annual meeting of the Texas Tuberculosis Association in Galveston on April 15-16. All officers were reelected for the 1956 term as follows: Mrs. Joella Terrill Butler, Wichita Falls, president; Arthur G. Schroeder, Victoria, first vice-president; Dr. Elliott Mendenhall, Dallas, second vice-president; J. W. Butler, Houston, secretary; Dr. Z. T. Scott, Austin, treasurer; and Dr. Robert B. Morrison, Austin, assistant treasurer. The 1956 meeting will be held April 6-7 in Midland.

### SOUTHERN MEDICAL ASSOCIATION

A kick-off dinner on March 19 in Houston signaled the beginning of plans for the forty-ninth annual meeting of the Southern Medical Association to be held in Houston, November 14-17. Attending the dinner were Drs. R. L. Sanders, Memphis, president; Milford O. Rouse, Dallas, chairman of the council; and V. O. Foster, Birmingham, executive secretary. Members of other medical organizations also were present, along with more than sixty physicians from Houston and Galveston.

### Central Texas Journal Club

The spring clinic of the Central Texas Journal Club held March 2 in Waco was dedicated to the late Dr. J. C. Bradford, Mart, who died last year while serving as vice-president of the Club.

# LIBRARY SECTION

## RECENT PUBLICATIONS BY TEXAS PHYSICIANS

Following is a list of recent articles published by Texas physicians and available at the Memorial Library of the Texas Medical Association. Articles appearing in the TEXAS STATE JOURNAL OF MEDICINE are not included. If a physician who has had a paper printed in the past six months is not named below, the Library would appreciate being notified. Also, the Library would like to have two reprints of each article.

Abbott, Jack P., and Fahlberg, Willson J.: Clinical Diagnosis of Fungous Disease, Postgrad. Med. *16:*319-329 (Oct.) 1954.

Ades, Harlow W.: Effects of Noise on Man, Dallas M. J. *40:*160-163 (Dec.) 1954.

Anthony, W. P.: Problem of the Chronically Discharging Ear, Bull. Tarrant County M. Soc. *26:*9-11 (Nov.) 1954.

Artz, C. P., and Howard, J. M.: Initial Care of the Severely Wounded, J.A.M.A. *156:*488-491 (Oct. 2) 1954.

Barnes, J. Peyton: Surgical Repair of a Large Omphalocele: Case Report, Am. Surgeon *20:*996-1002 (Sept.) 1954.

Bernard, Jack A.: Broad Spectrum Antibiotics, Southwestern Med. *35:*546-547 (Dec.) 1954.

Bernard, Jack A.: Gastrointestinal Antispasmodics, Southwestern Med. *36:*62 (Feb.) 1955.

Bernard, Jack A.: The Amebacides, Southwestern Med. *36:*111-112 (March) 1955.

Bernard, Jack A.: Parkinson Treatment, Southwestern Med. *26:*158 (April) 1955.

Blattner, R. J., and Heys, Florence M.: Host-Parasite Relationships in Virus and Rickettsial Disease: Approaches to Therapy, Postgrad. Med. *16:*270-281 (Oct.) 1954.

Blocker, T. G.; Levin, W. C.; Lewis, S. R.; and Snyder, C. C.: Use of Radioactive Sulphur Labeled Methionine in the Study of Protein Catabolism in Burn Patients, Ann. Surg. *140:*519-523 (Oct.) 1954.

Bloxsom, Allan: Newer Therapeutic Procedures Designed to Prevent Abnormal Pulmonary Ventilation in the Newborn Infant, J. Pediat. *45:*373-392 (Oct.) 1954.

Boyd, Charles D.: A Study of Flies and Their Ectoparasitic Mites as Related to the Epidemiology of Poliomyelitis, J. Tex. Pub. Health *6:*125-134 (Dec.) 1954.

Boylston, Bedford F.: Hemangioma of the Extremities, M. Rec. & Ann. *48:*78-82 (Oct.) 1954.

Braude, A. I.: Brucellosis: Epidemiology and Treatment, G. P. *11:*75-84 (March) 1955.

Brauer, Raymond O.: Report on My Experiences in Europe, Plast. & Reconstruct. Surg. *13:*307-316 (April) 1954.

Breck, Louis W.: Cysts of the Semilunar Cartilages of the Knee, Clin. Orthopedics No. 3, 1954, p. 29-38.

Brindley, G. V.: Treatment of Incurable Cancer of the Colon and Rectum, Surg., Gynec. & Obst. *100:*243-244 (Feb.) 1955.

Brindley, G. V., Jr.: Bronchogenic Carcinoma Simulating Benign Pulmonary Disease, A.M.A. Arch. Surg. *69:*740-745 (Nov.) 1954.

Browne, Frank S.: Benign Ulcer of the Greater Curvature of the Stomach, Am. J. Roentgenol. *73:*398-400 (March) 1955.

Bruce, E. Ivan, Jr.: Role of the Medical School in Psychiatry, Dis. Nerv. System *16:*61-63 (Feb.) 1955.

Brumage, William S.: We Know These Things About Cancer, Tex. Health Bull. *7:*12 (May) 1954.

Burdett, B. L.: Apparent Value of Vitamin $B_{12}$ in Treatment of Cerebral Vascular Spasm, Southwestern Med. *35:*515 (Nov.) 1954.

Calderon, Roberto; Ceballos, Jorge; and McGraw, John P.: Tomographic Aspect of Paralysis of the Vocal Cords, Radiology *63:*407-410 (Sept.) 1954.

Carman, H. Frank: Pleural Effusion, Postgrad. Med. *17:*203-204 (Feb.) 1955.

Chao, Dora; and Fields, William S.: Combined Drug Therapy in Petit Mal Epilepsy, J. Pediat. *45:*293-296 (Sept.) 1954.

Chapman, Eugene R.; Strozier, William E.; and Magee, Robert A.: Clinical Use of Apresoline in the Toxemias of Pregnancy, Am. J. Obst. & Gynec. *68:*1109-1117 (Oct.) 1954.

Clark, Helen, and Muirhead, E. E.: Plasmacytosis of Bone Marrow, Arch. Int. Med. *94:*425-432 (Sept.) 1954.

Clayton, R. S., and Goodman, Paul H.: The Roentgenographic Diagnosis of Geophagia (Dirt Eating), Am. J. Roentgenol. *73:*203-207 (Feb.) 1955.

Cobb, Beatrix; Clark, R. Lee, Jr.; McGuire, Carson; and Howe, C. D.: Patient-Responsible Delay of Treatment in Cancer, Cancer *7:*920-926 (Sept.) 1954.

Cody, Claude C., III: Surgical Anatomy of the Facial Nerve Outside the Skull, A.M.A. Arch. Otolaryng. *60:*291-301 (Sept.) 1954.

Cody, Claude C., III: Management of Usual Ear Diseases, Med. Rec. & Ann., *48:*115-122 (Nov.) 1954.

Coleman, S. D.: Observations on Tubal Pregnancies, Med. Rec. & Ann. *48:*123-124 (Nov.) 1954.

Colpitts, R. Vernon: Trends in Therapeutic Abortion, Am. J. Obst. & Gynec. *68:*988-997 (Oct.) 1954.

Cooley, Denton A.: Surgical Closure of Atrial Systal Defects, Surg., Gynec. & Obst. *100:*268-276 (March) 1955.

Cooley, Denton A., and DeBakey, Michael E.: Surgical Treatment of Mitral and Aortic Stenosis, J.A.M.A. *155:*235-239 (May 15) 1954.

Cooley, Denton A., and DeBakey, Michael E.: Resection of the Thoracic Aorta with Replacement by Homograft for Aneurysms and Constrictive Lesions, J. Thoracic Surg. *29:*66-104 (Jan.) 1955.

Cooley, Denton A., and DeBakey, Michael E.: Ruptured Aneurysms of Abdominal Aorta, Postgrad. Med. *16:*334-342 (Oct.) 1954.

Cox, E. Aubrey: Hidradenoma of the Perianum, Am. J. Surg. *88:*789-792 (Nov.) 1954.

Culotta, Ralph J.; Howard, John M.; and Johnson, Alice: Studies of Plasma Amylase in Acute Alcoholism, A.M.A. Arch. Surg. *169:*681-683 (Nov.) 1954.

Curtis, Dean D.: Naucaine Therapy in Nausea and Vomiting of Pregnancy, Obst. & Gynec. *5:*209-210 (Feb.) 1955.

Danzig, Lamont E., and Gomez, Alphonse C.: Long-Term Intravenous Use of Arfonad in the Treatment of Pulmonary Edema, Am. J. M. Sc. *228:*626-629 (Dec.) 1954.

DeBakey, M. E.; Cooley, D. A.; and Creech, Oscar: Aneurysms and Occlusive Diseases of the Aorta, G. P. *10:*52-57 (Nov.) 1954.

DeBakey, M. E.; Creech, Oscar, Jr.; Cooley, D. A.; and Halpert, B.: Structural Changes in Human Aortic Homografts, A.M.A. Arch. Surg. *69:*472-482 (Nov.) 1954.

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Denman, Frank R.: Transplantation Surgery for Arteriosclerotic Aneurysms and Occlusive Arterial Disease, M. Rec. & Ann. *48:*61-64 (Sept.) 1954.

Dennis, Edward: Treatment of Hypertension; Long Term Results with Alseraxglon (Rauweloid) Alone and Combined with Hexamethonium Administered Orally, Postgrad. Med. *16:*300-307 (Oct.) 1954.

Dennis, James L., and Hansen, Arild E.: Coccidioidomycosis in Children, Modern Med. *23:*134-138 (Jan. 15) 1955.

Dippel, A. Louis: Partial (Incomplete) Annular Detachment of the Cervix Uteri, Postgrad. Med. *16:*282-286 (Oct.) 1954.

Eads, R. B.; Von Zuben, F. S.; Bennett, S. E.; and Walker, O. L.: Studies on Cockroaches in a Municipal Sewerage System, Am. J. Trop. Med. *3:*1092-1098 (Nov.) 1954.

Ellison, Eugene T., and Thornton, W. D.: Late Results of Pelvic Surgery, South. M. J. *47:*913-918 (Oct.) 1954.

Etter, Richard L., and Merryman, George: Anaphylactic Shock and Death Due to Penicillin, Ann. Allergy *12:*453-454 (July-Aug.) 1954.

Fein, Bernard T.: Skin Tests in the Diagnosis of Internal Medical Disease, Am. Pract. & Digest Treat. *5:*897-904 (Dec.) 1954.

Fields, William S.: Practical Considerations in the Treatment of Epilepsy, Postgrad. Med. *16:*308-315 (Oct.) 1954.

Fish, Stewart A.: Complete Perineal Laceration, West. J. Surg. *62:*577-581 (Nov.) 1954.

Fish, Stewart A., and Shapiro, A. P.: New Drugs in Obstetrics and Gynecology, Dallas M. J. *41:*154-157 (March) 1955.

Fletcher, Gilbert H.; Old, Jacob W.; and Loquvam, G. S.: Topographic Approach to the Roentgenologic and Pathologic Examination of the Laryngopharyngeal Tumors, Radiology *63:*361-380 (Sept.) 1954.

Folbre, Thomas W.; Manhoff, Louis; and Frazier, Dean W.: Rhinoscleroma Successfully Treated with Oxytetracycline (Terramycin), A.M.A. Arch. Otolaryng. *60:*505-507 (Oct.) 1954.

Foraker, Alvan G.; Denham, Sam Wesley; and Mitchell, Dorothy D.: Histochemical Studies of the Effect of Irradiation of the Placenta, A.M.A. Arch. Path. *59:*82-89 (Jan.) 1955.

Ford, R. V., and Maley, N. S. R.: The Effect of Furadantin Therapy on Renal and Hepatic Function, J. Urol. *72:*959-962 (Nov.) 1954.

Fox, Everett C.: Herpes Gestationis (Dermatitis Herpetiformis): Case Report, A.M.A. Arch. Dermat. & Syph. *70:*331-335 (Sept.) 1954.

(This list will be continued in the next issue.)

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Deutschnerger, Otto: Fluoroscopy in Diagnostic Roentgenology, Philadelphia, W. B. Saunders, 1955.

Directory of Medical Specialists, vol. 7, Chicago, A. N. Marquis, 1955.

Forsee, James H.: Surgery of Pulmonary Tuberculosis, Philadelphia, Lea and Febiger, 1954.

Gairdner, Douglas: Recent Advances in Paediatrics, New York, Blakiston, 1954.

Goldsmith, W. Noel, and Hellier, Francis F.: Recent Advances in Dermatology, ed. 2, New York, Blakiston, 1954.

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## CONTRIBUTIONS TO THE LIBRARY

Grateful acknowledgment is made by the Texas Medical Association Memorial Library for the following recent gifts:

American Diseases of Children, 43 issues, no name received.

Dr. F. J. L. Blasingame, Wharton, 1,197 journals.

Dr. George E. Clark, Austin, 138 journals.

Dr. J. O. Holt, Jr., Dallas, 8 journals.

Dr. J. E. Johnson, Austin, 2 journals.

Dr. Sam N. Key, Sr., Austin, 6 bound journals.

Drs. Marion P. Primomo and John S. Primomo, Dilley, 125 journals.

Dr. N. L. Schiller, Austin, 24 journals, 22 reprints.

Dr. David R. Womack, Austin, 4 journals.

### Correction

The number of journals contributed to the Library in April by Dr. L. H. Reeves, Fort Worth, was incorrectly listed as 178 in the April issue of the JOURNAL. The correct number was 718.

## TAPE RECORDING FOR LOAN

### Dermatitis

Health Forum of the Travis County Medical Society, March, 1955. Tape recording for use on a standard tape recorder playing twin-track material at 3.75 inches per second. No. 12.

## MOTION PICTURES FOR LOAN

**A Life to Save**

*16 mm., sound, color, 27 minutes. Produced in 1954 for the American Medical Association. (Purchased by the Texas Medical Association Memorial Library.)*

This film is about middle-aged Mary Wilkens who is ill and on the advice of a neighbor goes to see a quack doctor in a nearby town. Mary's illness becomes worse with the quack's treatment. Fred Wilkens, Mary's husband, calls in the family doctor. The family doctor saves Mary's life, and with the cooperation of the county medical society and the American Medical Association the quack is brought to justice. This film is excellent for showing to all adult groups.

## BOOK NOTICES

**[1]Surgery of the Elbow**

FREDERICK M. SMITH, M. D., *Associate Professor of Clinical Orthopedic Surgery, Columbia University, New York. 340 pages. $10.75. Springfield, Ill., Charles C Thomas, 1954.*

The book gives a complete coverage of surgical conditions of the elbow. The approach to each subject is direct, but is of sufficient detail to make the subject clear. It should be recommended to any person dealing with injuries of the elbow joint, regardless of whether or not he may do surgery, since the preventive applications as well as the surgical applications are excellent.

**[2]Hysterectomy**

JOHN C. BURCH, M. D., *and* HORACE T. LAVELY, M. D. *94 pages. $5.50. Springfield, Ill., Charles C Thomas, 1954.*

This is an excellent monograph and particularly so since in the past few years there has been much discussion about indications for removal of the uterus and about unnecessary pelvic surgery.

Chapter one, entitled "The Hysterectomy Problem," discusses the number of unnecessary hysterectomies done and may be summed up under the title of Dr. Norman Miller's paper, "Hysterectomy, Therapeutic Necessity or Surgical Racket."

The second chapter, entitled "An Approach to Hysterectomy," reveals the harm done by unnecessary hysterectomies and gives a good discussion of the conservation of the uterus at the time of surgery when this organ is useless and can result only in future disease. The psychologic approach is also discussed.

Chapter three deals with some conditions requiring hysterectomy, and here the authors carefully weigh the pro's and con's of each indication.

Chapter four deals with the choice of operation and gives indications for abdominal and vaginal approach.

Chapter five, parts A, B, and C, deal with the technique of abdominal hysterectomy, vaginal hysterectomy, and radical hysterectomy. These are beautifully illustrated with hand-drawn illustrations by Helen Lorraine.

Chapter six deals with intra-operative complications such as hemorrhage and bladder, ureteral, and bowel injuries and discusses the treatment of these complications. Here also is discussed the choice of hysterectomy clamps and ligatures.

The authors report 1,226 hysterectomies of all types without a death.

The physiologic effects of hysterectomy are summed up as follows:

1. The mortality varies from 2 to 5 deaths per 1,000 operations.
2. The saving of life in terms of cancer prevention is 25 lives per 1,000 cases in white persons and 35 per 1,000 in Negro patients between the ages of 35 and 45 years.
3. The functions of reproduction and menstruation are removed with the uterus.
4. Ovarian function continues at or about the same level.
5. Sexual function is either improved or unchanged.

This is an excellent monograph.

**[3]Lesions of the Lumbar Intervertebral Disc**

R. GLEN SPURLING, *Clinical Professor of Surgery (Neurosurgery), University of Louisville School of Medicine, Louisville, Ky. 148 pages. $4.50. Springfield, Ill., Charles C Thomas, 1953.*

The embryologic and anatomic considerations are discussed. This is followed by consideration of the pathogenesis and pathologic process of lumbar intervertebral disc protrusions. The clinical picture, diagnosis, and roentgenologic diagnosis are then described, and this is followed by a chapter on therapy and finally a chapter on the results of therapy. The author in this excellent monograph has presented in a clear and concise manner the pertinent facts relating to lumbar intervertebral disc lesions. This monograph should be of particular interest to the general practitioner as it will provide him with an excellent working knowledge of this disease.

**[4]Arrest of Bleeding**

JACQUES ROSKAM, *Professor of Internal Medicine, University of Liege, Belgium. 71 pages. $2.75. Springfield, Ill., Charles C Thomas, 1954.*

A detailed analytical study of spontaneous hemostasis is presented. Facts of this study disclose that hemostasis from small arteries and veins depends upon formation of a white thrombus or hemostatic plug. Capillary bleeding ceases by blood coagulation if bleeding is not prevented by adherence of endothelial cells.

The bleeding time, though not reliable in the detection of major hemorrhagic diseases, is useful in the study of hemostasis. The "mean bleeding time" is most reliable and consists of an average of a number of individual bleeding times from different areas of the ears under constant conditions. Using the mean bleeding time, physiologic studies reveal that a combined action of two or more factors are necessary for uncontrollable hemorrhages. A study of various agents shows that some vasoconstrictors and many so-called hemostatic drugs do not shorten bleeding time. A detailed explanation of each is given. A new hemostatic, adrenochrome, is proven to be a physiologically acting agent whereas other drugs seem to reduce bleeding by their quinonic functions. Adrenoxyl (monosemicarbazone of adrenochrome) is prophylactic but does not modify hemorrhage which precedes its administration. Naphthionine SNS and adrenalone or stryphnon have a delayed hemostatic action. Calcium derivatives favor hemostasis both orally and parenterally. Blood coagulants such as coagulen, sangostop, coagucit, and small doses of thrombin improve spontaneous hemostasis. When the direct control of bleeding is impossible and the blood picture does not show definite hemorrhagic disease, good chances for success in treatment may be had by the "omnibus" method: (1) a vascular

[1]*Sterling M. Hardt, M. D., Bastrop.*
[2]*George F. Adam, M. D., Houston.*
[3]*Albert A. LaLonde, M. D., Austin.*
[4]*L. T. Green, Jr., M. D., Muleshoe.*

hemostatic such as adrenoxyl, stryphnon, or naphthionine SNS, (2) a coagulant such as thrombin, coagulen, or sangostop, and (3) any calcium derivative (mechanism of action still unknown).

Experiments with heparin prove that prolonged bleeding depends upon the combined action of at least two hemorrhagic factors, one being vascular, the other consisting of some disorder of blood itself.

**[5]A Formulary for External Therapy of the Skin**

CHESTER N. FRAZIER, M. D., Dr. P.H., *Edward Wigglesworth Professor of Dermatology, Harvard Medical School; Chief of the Dermatological Service, Massachusetts General Hospital, Boston, and* IRVIN H. BLANK, Ph. D., *Research Associate in Dermatology, Harvard Medical School, Massachusetts General Hospital, Boston. 118 pages. $3.25. Springfield, Ill., Charles C Thomas, 1954.*

This is an excellent treatise on external therapy of the skin. Those who are looking for a long list of prescriptions or formulas for the treatment of skin diseases will be disappointed as this book deals mostly with the principles of external therapy. It should be required reading for every undergraduate student of dermatology, and the experienced dermatologist will find much of interest and value in it.

**[6]Clinical Aspects of the Autonomic Nervous System**

L. A. GILLILAN, Ph. D., M. D., *Associate Professor of Anatomy, Graduate School of Medicine, University of Pennsylvania. 316 pages. $6.50. Boston, Little, Brown and Company, 1954.*

The title of this book is somewhat misleading, as it concerns itself almost entirely with anatomy. The author has done a workman-like job of describing the complex connections of the autonomic nervous system, using original diagrams in a helpful manner. In regard to clinical applications he has treated very complex subjects in a casual fashion, making dogmatic statements that are at least controversial in current thinking. The book is best suited to furnishing the anatomic background for the understanding of autonomic dysfunction.

**[7]Peripheral Vascular Diseases**

EDGAR V. ALLEN, M. D., M. S. in Med., F.A.C.P., *Section of Medicine, Mayo Clinic; Professor of Medicine, Mayo Foundation, Graduate School, University of Minnesota;* NELSON W. BARKER, M. D., M. S. in Med., F.A.C.P., *Section of Medicine, Mayo Clinic; Professor of Medicine, Mayo Foundation, Graduate School, University of Minnesota; and* EDGAR A. HINES, JR., M. D., M. S. in Med., *Mayo Clinic; Professor of Medicine, Mayo Foundation, Graduate School, University of Minnesota. ed. 2. 825 pages. $13. Philadelphia, W. B. Saunders, 1955.*

The recent increase in incidence and clinical importance of peripheral vascular disease plus the revolutionary changes in therapy make a new book in this field most welcome. The second edition of "Peripheral Vascular Disease" is such a book. It is a modern, encyclopedic work based largely on the vast personal experience of the authors and their associates at the Mayo Clinic and a broad knowledge of the literature. The subject matter includes diseases of "all of the vessels distal to the heart" as well as those of the lymph vessels. Arterial hypertension and vascular diseases of the central nervous system and some of the viscera are omitted. Practical, clinical applications are always stressed. Controversial subjects are frankly and clearly discussed in the light of the authors' experience and observations; defects in present knowledge are noted and suggestions for future research are presented.

The revision of the second edition has been thorough and has incorporated the best of the many advances in this dynamic field which have appeared since the first edition in 1946. Some of the material dealing with special methods of study has been deleted. The authors have added entirely new presentations of aortography; coarctation of the aorta; purpura; hypertensive ischemic ulcers of the legs; techniques of lumbar sympathectomy; nonvascular operations for intermittent claudication; and surgical treatment of varices, aortic aneurysms, and vascular injuries. The section on anticoagulants has been entirely rewritten. The chapters on "Medical Treatment" and "Surgical Treatment" are well done and reflect the current practices of the Mayo Clinic. This book is as new as January, 1955.

The subject matter is well organized and integrated for easy reference. The book is convenient to hold, well bound and printed. There is a full table of contents; a brief outline at the beginning of each chapter; an adequate bibliography at the end of each chapter; and a complete index. The 316 illustrations are clear, well chosen, and strategically located in the text. The style is easy to read and conversational.

The book has two goals: "to aid physicians who must care for patients with peripheral vascular disease and to provide information for the student who is more interested in historical development, physiology, pathology, and methods of investigation." The authors have succeeded well in their goals. They have produced the most complete, up-to-date reference and working manual available today on this subject.

**[8]Practice of Allergy**

WARREN T. VAUGHAN, M. D., *Richmond, Va. Revised by* J. HARVEY BLACK, M. D., *Dallas. Ed. 3. 1,164 pages. $21. St. Louis, C. V. Mosby Company, 1954.*

That this book has gone into its third edition is testimony of its value in the allergic field. Although allergy and immunology is a relatively young field of medicine, an enormous amount of literature has accumulated about it, and there have been many changes in thinking about it—all covered in this third edition.

The first two parts cover the history, theory, and physiology of allergy. The remainder of the book completely discusses the diagnosis and treatment of clinical allergy. Sections are included on the pollens of the various parts of the country. The section on foods is a good tabulation of all the various foods causing allergy. In spite of the thor oughness and completeness of this work these authors point out that there is still much more research to be done and much more to be learned of allergy and its manifestations.

This book should be included in the library of the serious student of allergy.

**[9]Public Relations in Medical Practice**

JAMES E. BRYAN, *Administrator, Medical-Surgical Plan of New Jersey. 301 pages. $5. Baltimore, Williams and Wilkins Company, 1954.*

Mr. Bryan has done a valuable service for the medical profession in writing this book concerning the public relations of physicians. There is no question that the public relations of the medical profession can stand much improvement at the present time. The author has a readable style that holds one's interest like a storybook. Mr. Bryan,

[5]*Maurice C. Barnes, M. D., Waco.*
[6]*James W. Lassiter, M. D., Austin.*
[7]*J. Lawrence Clark, M. D., Ennis.*
[8]*A. M. Allison, M. D., Alice.*
[9]*William M. Crawford, M. D., Fort Worth.*

though not a physician, has been closely connected with the medical profession as executive secretary of a county medical society and of a state medical society and as an administrator of a Blue Shield plan over a period of more than twenty years. He is well qualified to write on this subject.

The book is composed of 301 pages in large readable type and can easily be read in an evening. The book also will be valuable as a reference work on this subject. Public relations is thoroughly covered as can be determined by the chapter headings: "The Doctor and His Patient," "The Doctor and His Fee," "The Doctor and His Office Staff," "The Doctor and His Colleagues," "The Doctor and His Community," "The Doctor and His Medical Society," "The Doctor and Prepaid Medical Care," "The Doctor and His Hospital," and "The Doctor and Himself."

It is agreed that few physicians get enough "indoctrination" into the practice of medicine after leaving their internships. This book can certainly fill this gap for the young physician entering into the practice of medicine. There is also much timely information which it would pay our older members to think about and review.

The physicians' ethical code is composed of the Hippocratic Oath, the American Medical Association Code of Ethics, and the Golden Rule. Medical care has changed both in its manner of distribution and mode of payment; however, as this book shows, it has not changed our primary thesis of "service to humanity" and the "preservation of life."

There is an interesting, thought provoking foreword by Louis H. Bauer, Past President of the AMA, that is timely.

This book should be required reading for all young physicians upon joining their respective medical societies and also a valuable addition to the older doctor's library for crystallizing present day thought on a perplexing problem that must be faced by members of the medical profession. I heartily recommend this book for all physicians.

**[10]An Introduction to Physics in Nursing**

HESSEL HOWARD FLITTER, R. N., M. A., *Assistant Professor and Director of Science Instruction, School of Nursing, University of Pennsylvania; Instructor of Physics Applied to Nursing, New York University, School of Education. ed. 2. 208 pages. $3.50. St. Louis, C. V. Mosby Company, 1954.*

This represents a compactly presented resume of elementary physics as it pertains to commonly used procedures and instruments in a hospital. For example, the principles of work, energy, and power, of wheels, levers, inclines, screws, jacks, and pulleys are explained and illustrated. The physiologic relationship of physical principles is considered where appropriate, as for example, the principles of fluid pressure as they relate to the cerebrospinal fluid, blood, and the urinary bladder. In this connection the practical aspects of administering intravenous fluids are covered with discussions of pressure gradient, friction, head of fluid, viscosity of fluid, and laminar and turbulent flow, as well as Poiseuille's law with mathematical formulas summarizing the factors involved in fluid flow.

Included are discussions of the present status of radioactive substances used clinically so that the student should be well acquainted with what they are, but the book gives few instructions or do's and don't's as related to clinical nursing care of patients on whom such substances are used. The presentation of electricity and electronics is elementary but good.

One is favorably impressed by this effort, and it is probable that it would be a valuable book in the library of any nursing school.

[10]*Joe C. Rude, M. D., Austin.*

# ORGANIZATION SECTION

## AMERICAN MEDICAL ASSOCIATION

### AMA Atlantic City Meeting

Between 13,000 and 16,000 physicians are expected to attend the one hundred fourth annual session of the American Medical Association in Atlantic City, N. J., June 6-10. Scientific programs including reports of interest to physicians in nearly every field of medicine will be presented, and a new feature, "Queries and Minor Notes," will assure that consultants in every branch of medicine will be on hand in Convention Hall to discuss and answer doctors' questions concerning specific cases.

More than 325 scientific exhibits and 350 technical exhibits will be on display in the Atlantic City Auditorium, which will be open from 8:30 a. m. until 5:30 p. m. Monday through Thursday, and from 8:30 a. m. until noon on Friday. Among the exhibits will be one depicting the fiftieth anniversary of organized anesthesia, an exhibit on pulmonary function testing, and a fresh tissue pathology exhibit. The Air Force will demonstrate its "flying infirmary" on the beach in front of the Convention Hall.

The House of Delegates will convene on Monday morning, June 6, and the Scientific Assembly will open at 9 a. m. the same day with the General Scientific Meetings. The Inaugural Meeting at which Dr. Elmer Hess, Erie, Pa., will be installed as President of the American Medical Association, will be broadcast nationwide on Tuesday evening, June 7. The guest speaker will be Norman Vincent Peale, D. D., pastor of the Marble Collegiate Church of New York.

For the benefit of physicians unable to attend the convention, the National Broadcasting Company's television network will televise on June 7 a show beamed directly from the Convention Hall. It will be the third program in the spring series of the "March of Medicine."

Interesting surgical and clinical demonstrations will be televised in color and will be piped directly into the Auditorium from Philadelphia hospitals.

Registration for the five-day meeting will be held in Convention Hall on the Boardwalk. An information bureau will be operated in connection with the Registration Bureau. General officers of the AMA and members of the House of Delegates may register for the Scientific Assembly at the entrance to the American Room at the Hotel Traymore.

In addition to the full program of scientific programs and exhibits, there will be a reception and ball for the President after the inaugural ceremony June 7. The American Medical Golfing Association will hold its tournament at the Atlantic City Country Club June 6, and fraternity, alumni, and other organizations have scheduled meetings and luncheons during the session. Members of the Woman's Auxiliary will meet at the Haddon Hall Hotel for general meetings, and several social functions have been planned.

For physicians who wish to take vacations either immediately before or after the convention, special tours of Europe are on the agenda, plus a post-convention cruise to Bermuda and Nassau.

Information regarding hotel reservations may be obtained by writing the American Medical Association, Chairman, Subcommittee on Hotels, 16 Central Pier, Atlantic City, N. J.

### Boston Clinical Meeting

The Boston Clinical Meeting of the American Medical Association will meet in Boston November 29-December 2. Persons who wish to speak on the lecture program may write to Dr. Theodore L. Badger, chairman of the program committee, Massachusetts Medical Society, 22 The Fenway, Boston 15.

## TEXAS MEDICAL ASSOCIATION

### DOES YOUR COMMUNITY NEED A DOCTOR?

Does your community need a doctor? The Texas Medical Association, through the combined efforts of its Physicians Placement Service and Committee on Rural Health and Doctor Distribution, has taken the initiative among the state associations toward finding an answer to this simple, yet complex, question.

Following in the footsteps of separate progress by the Physicians Placement Service and the doctor distribution surveys, reported in the TEXAS STATE JOURNAL OF MEDICINE of February, 1954, the joining together of these two important efforts established the Texas Medical Association as the first constituent state organization of the American Medical Association to use a community rating schedule.

Knowing full well that the community rating schedule designed by the Council on Medical Service of the AMA was an experimental program, the Association, on April 7, put it into operation in the hope that it might serve several purposes: (1) to stimulate communities to analyze their characteristics and facilities from a physician's viewpoint; (2) to ascertain the extent of the minimum amount of information needed on each opening; (3) to supply to the AMA and other state associations valuable information and differentiation among the communities listed as needing a physician.

In laying the yardstick of medical need and desirability beside a particular community, the primary objectives were (1) to determine where a need for a physician actually exists and estimate the degree of that need; (2) to evaluate the medical desirability of a given location; (3) to assist communities in improving their medical desirability rating; (4) to guide physicians in their choice of locations.

The first step taken was to designate who was to serve on a survey team. After much deliberation, it was decided that, in acting on any particular request for the services of the community rating survey team, the team should be composed of the Councilor of the district, an officer of the local county medical society, the field director of the Association, and the coordinator of the Physicians Placement Service in the central office.

During the past month such a team has met with the key civic leaders of three communities, Brackettville, Sabinal, and Stockdale, to conduct a community rating. Since this effort was the first of its kind, the team found it necessary to move slowly and evaluate carefully in order that it might draw sound recommendations and conclusions.

From the standpoint of community need, calculations were based and ratings made on (1) doctor-population ratio; (2) distance to nearest adequate medical care; (3) age of physicians serving the area; (4) intangible factors, such as public expression of need and the community's willingness to back its request with action.

Desirability priority, from the standpoint of the physician, was based and rated on (1) hospital facilities accessible; (2) office facilities offered by the community; (3) opportunities for assured income other than regular practice; (4) facilities for family life, including housing, schools, churches, and social opportunities; (5) intangible factors, such as availability of diagnostic service, ambulances, nursing service, and registered pharmacists.

The survey team found exhaustive observation and questioning to be a vital requisite in obtaining this information. It was gratifying to note that the citizens of these communities laid all their cards on the table (including those considered detrimental) and gave the survey team honest, forthright answers to all questions.

The community rating survey team was well received in those communities surveyed. In addition to the invaluable information gathered for the benefit of the Association, this program proved to be an important public relations link between the medical profession and the public. Without exception, every member of each local committee voiced praise of this program and stated that this was their first realization that the Texas Medical Association served anybody other than the physicians. A further, and perhaps more important, observation was that they had never fully understood the physicians' side of medical care when viewed from the proper perspective on such matters as its adequacy, economics, and distribution.

The progress of this program was noted at the annual session by the House of Delegates with a vote of confidence and a recommendation that these efforts be continued throughout the coming year. This activity is one of the many tools employed by the Texas Medical Association to bring about a closer liaison of thought and understanding between the private citizen and the medical profession.

## COUNTY SOCIETIES

### Bastrop-Lee Counties Society

February 22, 1955

At a joint meeting with the Woman's Auxiliary, the Bastrop-Lee Counties Medical Society met at the home of Dr. and Mrs. Albert F. Vickers. Harvey Renger, Hallettsville, chairman of the Texas Medical Association's Council on Medical Economics, spoke to the group.

### Bell County Society

April 6, 1955

(Reported by J. B. Brown, Secretary)

The Bell County Medical Society met in Temple on April 6. Methods of handling inoculation of first and second graders with the Salk vaccine were discussed, and a public relations committee was appointed. The guest speaker, Lt. Col. Frank M. Townsend, Galveston, discussed the Air Force medical research and development program.

### Borden-Dickens-Garza-Kent-King-Scurry-Stonewall Counties Society

April 6, 1955

F. J. L. Blasingame, President of the Texas Medical Association, was honored at a dinner in Snyder given by the Borden-Dickens-Garza-Kent-King-Scurry-Stonewall Counties Medical Society on April 6.

### Bowie-Miller Counties Society

March 10, 1955

The Physician's Stake in National and State Legislation—Milford O. Rouse, Dallas.

The Bowie-Miller Counties Medical Society met in Tex-

arkana on March 10. Dr. Rouse, head of the Texas Delegates to the American Medical Association, gave the above address.

### Brazoria County Society

April 2, 1955

C. H. Kim, Seoul, Korea; J. Boss, London, England; and W. Hild, Germany, graduate students at the University of Texas Medical Branch, spoke on the various phases of medicine in their countries at the April 2 meeting of the Brazoria County Medical Society in Freeport. The meeting was a joint one with the Woman's Auxiliary.

### Cameron-Willacy Counties Society

February 21, 1955

Michael DeBakey, Houston, presented a technical paper concerning surgical procedure to members of the Cameron-Willacy Counties Medical Society at the February 21 meeting in Brownsville.

March 21, 1955

Tuberculosis and Its Modern Treatment—David McCullough, Kerrville.

The March 21 meeting of the Cameron-Willacy Counties Society was held with sixty members and guests present. The above scientific program was presented, after which a report on the proposed opening of the tuberculosis hospital in September was given.

### Cass-Marion Counties Society

March 23, 1955

Bronchial Asthma—Andrew Goesl, Texarkana.

The Cass-Marion Counties Medical Society met in Jefferson on March 23 and heard the above mentioned program. Members of the Woman's Auxiliary were dinner guests.

### Colorado-Fayette County Society

April 5, 1955

Following a steak dinner, the Colorado-Fayette County Medical Society met on April 5 in Schulenburg. Twenty-two members were present.

### Dallam-Hartley-Sherman-Moore Counties Society

March 3, 1955

Dalhart was the site of the March 3 meeting of the Dallam-Hartley-Sherman-Moore Counties Medical Society. Guest speaker was Mr. C. Lincoln Williston, Austin, Executive Secretary of the Texas Medical Association, and H. H. Latson and E. A. Rowley, both of Amarillo.

### Dallas County Society

March 8, 1955

(Reported by Glenn D. Carlson, Secretary)

Newer Drugs in Treatment of Epilepsy—Stephen Weisz, Dallas.
Surgical Treatment of Epilepsy—Grant L. Boland, Dallas.
Emotional Aspects in Epilepsy—James K. Peden, Dallas.

Members of the Dallas County Medical Society met on March 8 at the Baylor Hospital and were dinner guests of the Baylor Medical Staff. Following the above scientific program, a business meeting was held, and ten new members were elected.

### Eastland-Callahan-Stephens-Shackelford-Throckmorton Counties Society

March 15, 1955

Oral Malignancies—Burgess Sealey, Fort Worth.
Pathology of Oral Malignancies—C. T. Ashworth, Fort Worth.

The above scientific reports were presented to the Eastland-Callahan-Stephens-Shackelford-Throckmorton Counties Medical Society at a March 15 dinner meeting in Ranger. Other guests were R. G. Baker, Fort Worth, district councilor, and the following members of the Texas Medical Association central office staff in Austin: Mr. C. Lincoln Williston, Executive Secretary; Mr. J. Stuart Page, field director; and Mr. Dan Lehman, coordinator of exhibits.

### Falls County Society

(Reported by Walter L. Reese, Secretary)

The 1955 officers of Falls County Medical Society are James Mitchell Brown, president; Douglas R. Swetland, vice-president; Walter L. Reese, secretary-treasurer; Neil D. Buie, delegate; and Alfred C. Bennett, alternate delegate. All are from Marlin.

### Galveston County Society

March 24, 1955

The Galveston County Medical Society voted to support the administering of the Salk vaccine in Galveston County at a meeting on March 24 in Galveston.

### Hale-Floyd-Briscoe Counties Society

March, 1955

Plans were laid at the March meeting of the Hale-Floyd-Briscoe Counties Society for the inoculation of all eligible public school children in that area. A committee was appointed to map a tentative program.

### Harrison County Society

February 17, 1955

The Harrison County Medical Society met February 17 in Marshall and was host to Dr. and Mrs. F. J. L. Blasingame, Wharton, and Mr. C. Lincoln Williston, Austin. Dr. Blasingame, President of the Texas Medical Association, addressed the group on "Socio-Economic Trends and Their Influence on Private Practice." Mr. Williston, Executive Secretary of the Association, spoke about the various services the Association is offering members.

Members of the Cass-Marion and Upshur Counties Societies and their wives were invited to attend.

### Hidalgo-Starr Counties Society

March 11, 1955

The Hidalgo-Starr Counties Medical Society heard a report on the latest developments in the use of the Salk vaccine at its March 11 meeting in Pharr.

### Hill County Society

March 22, 1955

G. V. Brindley, Jr., Temple, spoke at the joint annual dinner meeting of the Hill County Medical Society and Hill County Tuberculosis Association in Hillsboro on March 22. He traced the history of tuberculosis treatment and explained the modern methods of treatment.

### Howard-Martin-Glasscock Counties Society

March 22, 1955

Recent Theories of Blood Coagulation—Captain J. F. Johnson, Webb Air Force Base.
Arfonad—Its Use to Control Hypotension (motion picture).

The March 22 meeting of the Howard-Martin-Glasscock Counties Medical Society was held at Webb Air Force Base, Big Spring. Members of the society were invited as guests of the hospital personnel. Following a dinner, the program was presented as outlined above.

April 19, 1955

What the General Practitioner Should Know About Glaucoma—J. H. Fish, Big Spring.
Glaucoma (motion picture)—Dr. Fish.

Following the above program of the April 19 meeting of the Howard-Martin-Glasscock Counties Medical Society,

a business meeting was held at which members discussed a general policy of poliomyelitis immunization of private patients. Their decision on policy was forwarded to the local newspapers.

### Hunt County Society

April, 1955

At its meeting early in April, the Hunt County Medical Society voted to go on record as approving the establishment of a county hospital in Greenville.

### Lamar County Society

April 7, 1955

(Reported by Harold E. Hunt, Secretary)

Rectal Carcinoma with Fistulation into Bladder — George Woodfin and Donald Lewis, Paris.

Twenty-one members and five guests of the Lamar County Medical Society attended the dinner meeting in Paris on April 7. The business meeting was centered around plans for the Salk vaccine inoculations.

### Nueces County Society

March 8, 1955

F. J. L. Blasingame, Wharton, President of the Texas Medical Association, spoke at a joint meeting of the Nueces County Medical Society and its Woman's Auxiliary in Corpus Christi on March 8. He said that doctors must sell private practice to the public.

### Pecos-Jeff Davis-Presidio-Brewester Counties Society

April 5, 1955

Recent Developments in Cardiovascular Disease—Jack C. Postlewaite, El Paso.

Jack C. Postlewaite, internist from El Paso, spoke before the Pecos-Jeff-Davis-Presidio-Brewester Counties Medical Society meeting in Marfa April 5.

### Tarrant County Society

March 15, 1955

(Reported by S. W. Wilson, Secretary)

Panel Discussion: Liver and Biliary Dysfunctions — James A. Farley, moderator; Warren W. Moorman, Charles Robinson, and T. J. Coleman, all of Fort Worth, panel members.

The Tarrant County Medical Society met March 15 in Fort Worth and heard the above mentioned panel discussion. A resolution that the American Medical Association be urged to establish a plan for accreditation of hospitals and that the American Hospital Association be urged and invited to cooperate, was adopted. The society also resolved to approve the article to the United States Constitution as proposed in House Concurrent Resolution 63, which would prevent the government from engaging in businesses or professions except those specified in the Constitution.

April 5, 1955

(Reported by S. W. Wilson, Secretary)

Pediatric Urology—Harry Spence, Dallas.

The indicated scientific program was given at the April 5 meeting of the Tarrant County Medical Society in Fort Worth. The society went on record as opposing the President's Health Program and the bills introduced in Congress which would make compulsory Social Security for any groups of the self-employed which are now exempt from the tax.

April 19, 1955

(Reported by S. W. Wilson, Secretary)

Tumors of the Neck—Paul M. Ramey, Temple.

At the April 19 meeting of the Tarrant County Medical Society, William M. Crawford spoke regarding the coming meeting of the Texas Medical Association. Mr. C. Lincoln Williston, Executive Secretary of the Association, thanked the society and members for the support and efforts given in connection with the convention. The program was given as outlined above.

### Travis County Society

April 19, 1955

(Reported by C. H. McCuistion, Secretary)

The Travis County Medical Society met on April 19 in Austin. After a short business session regarding the cancer program, there was a round-table discussion on immunization.

### Washington-Burleson Counties Society

March, 1955

At its meeting in Brenham early in March, the Washington-Burleson Counties Medical Society voted to administer the Salk vaccine free of charge to first and second grade children, pending the final approval of the vaccine.

### Wharton-Jackson-Matagorda-Fort Bend Counties Society

April 12, 1955

(Reported by Stanley E. Thompson, Secretary)

Mr. W. E. Syers, Austin, public relations counsel of the Texas Medical Association, spoke on the business side of medical practice at the April 12 meeting of the Wharton-Jackson-Matagorda-Fort Bend Counties Medical Society in Wharton. Dr. F. J. L. Blasingame, Wharton, President of the Texas Medical Association, talked about the responsibilities of the members of the county societies and their relation to the Association and the American Medical Association. Mr. J. Stuart Page, Austin, field director of the Association, told of its functions and services.

## DISTRICT SOCIETIES

### Second District Society

April 21, 1955

(Reported by M. J. Loring, Secretary)

Clinical Course and Management of Acute Renal Failure—E. E. Muirhead, Dallas.
Human Equation in Plastic Surgery—John F. Pick, Chicago.
Surgical Abrasions of the Face (motion picture)—John F. Pick.
Lesions of the Central Nervous System as Seen by the Practitioner: Part One, Brain Lesions; Part Two, Spinal Cord, Nerve Roots, and Peripheral Nerves—Grant L. Boland, Dallas.
General Survey of Hemorrhagic Diseases—Dr. Muirhead.

The Second District Medical Society met on April 21 in Midland. After the above scientific program and a business meeting, a social hour for the doctors and their wives was held followed by a dinner. New officers are T. W. Novak, Odessa, president; John H. Fish, Big Spring, vice-president; and Willis T. Carson, Odessa, secretary-treasurer. The next meeting will be held April 19, 1956, in Odessa.

### Third District Society

April 13, 1955

Complications and Nonspecific Treatment of Hypotension—Ben J. Wilson, Dallas.
Modern Concept of Surgical Correction of Protruding Ears—Ben T. Withers, Houston.
Management of Obstetrical Emergencies During First Three Stages of Labor—Isadore Dyer, New Orleans.
Ulcerative Disease of Upper Gastrointestinal Tract—Gordon McHardy, New Orleans.

The District 3 Medical Society met in Borger on April 13, and the above scientific program was presented at the morning session. After a luncheon and brief business meeting, the session was divided into four special interest groups,

eye, ear, nose, and throat; medical; surgical; and obstetrics and gynecology. Specialized scientific programs were presented at each of the sections. The meeting was climaxed by a cocktail party and buffet dinner and dance.

### Eleventh District Society

March 31, 1955

(Reported by Hugh F. Rives, Secretary)

Technique and Review of Upper Gastrointestinal Roentgen-Ray Studies —Floyd G. Betts, Tyler.

Technique and Review of Barium Enema Roentgen-Ray Studies—G. W. Bilbro, Jacksonville.

Use of Hypnosis in Surgical Dressings—Morris J. Fogelman and H. B. Crasilneck, Ph. D., Dallas.

Clinical Pathological Conference—Harbert Davenport, Jacksonville.

Twenty-nine members attended the Eleventh District Medical Society's March 31 meeting in Athens. The above outlined program was presented, and was followed by a business meeting and a dinner at the Athens Country Club for the members and their wives and guests. The fall meeting will be held in Palestine.

### Fifteenth District Society

April 14, 1955

The Fifteenth District Medical Society met on April 14 in Mount Pleasant and elected James H. Harris, Marshall, president; G. A. Farquhar, Marshall, vice-president; and L. E. Rutledge, Daingerfield, secretary-treasurer. J. Layton Cochran, President-Elect of the Texas Medical Association, and Mr. C. Lincoln Williston, Executive Secretary of the Association, were guests. The 1956 meeting will be held in Marshall.

# AUXILIARY SECTION

## COUNTY AUXILIARIES

Members of the *Bexar County Auxiliary* staged an exhibit, "Human Diseases Transmitted from Animals to Man," at the Livestock Exposition in San Antonio during the month of February. Co-chairmen of the project were Mesdames John C. Parsons and Everett T. Duncan.

The *Denton County Auxiliary* and the *Brazoria County Auxiliary* have established scholarships for deserving young women who wish to enter nurses training. In another area of educational support, the *Grayson County Auxiliary* presented proceeds from a March 4 style show to the American Medical Education Foundation fund. Voluntary contributions and memorial gifts make up the *Dallas County Auxiliary's* donation to the AMEF drive and the Memorial Fund. Early in March, the Dallas group also gave two rare medical books to the Memorial Library of the Texas Medical Association.

Mrs. Mark H. Latimer, Houston, President of the Woman's Auxiliary to the Texas Medical Association, has been the guest of several county auxiliaries recently. In Sulphur Springs, Mrs. Latimer was honored with a luncheon at the March meeting of the *Hopkins-Franklin Counties Auxiliary*, where she spoke on "Better Health in a Free America." As the guest of the *Henderson County Auxiliary* at its March meeting, Mrs. Latimer spoke informally about matters concerning the medical profession and the part auxiliaries can play in public relations and health programs. The meeting was in the home of Mrs. J. W. Gibson, Athens, who is the new president of the group. Other officers are Mesdames Don Price, vice-president, and J. H. Burtis, secretary-treasurer.

Emphasizing the shortage of nurses, Mrs. Latimer spoke to members of the *Howard-Martin-Glasscock Counties Auxiliary* at a tea at the home of Mrs. P. W. Malone, Big Spring, early in February. Several days later, she gave members of the *Andrews-Ector-Midland Counties Auxiliary* a resume of the history and goals of the Woman's Auxiliary at the meeting in Midland, where Mesdames Harold Lindley, Pecos, second vice-president of the State Auxiliary, and August J. Streit, Amarillo, fifth vice-president, also were guests. Briefly reviewing the work of the Auxiliary throughout the state, Mrs. Latimer spoke to the *Cherokee County Auxiliary* at a luncheon February 22 at the home of Mrs. M. L. Gray, Jacksonville. New officers of the Cherokee auxiliary are Mrs. J. M. Travis, president; Mrs. Hugh E. Rives, vice-president; Mrs. G. W. Bilbro, corresponding secretary and treasurer; and Mrs. Don Martin, recording secretary.

Joe Z. Tower, Ph.D., was guest speaker at the *Angelina County Auxiliary* meeting February 22. His topic was "Why Our Children Are Delinquent." At the March 11 meeting of the *Bexar County Auxiliary*, in San Antonio, Maj. Gen. James P. Cooney spoke on the medical aspects of atomic energy. A speech on civic enterprise was presented to members of the *Dallas County Auxiliary* by Charles R. Meeker, Jr., managing director of the State Fair Musicals, at their March 2 meeting in Dallas.

"The New Woman," a talk by Andrew Edington, Ph. D., president of Schreiner Institute, was presented to members of the *Kerr-Kendall-Gillespie-Bandera Counties Auxiliary* at their meeting March 4 in Kerrville. Dr. Henry A. Holle, Texas state health officer, was guest speaker at the Austin meeting of the *Travis County Auxiliary* on February 15. Dr. Charles Collins, Wichita Falls State Hospital, spoke on mental health to the ladies of the *Wichita County Auxiliary* at a luncheon March 8. A discussion of President Eisenhower's health program for 1955 was sparked by Mrs. Carlton E. Wolters at the *Harris County Auxiliary's* meeting in Houston on February 28.

Members of the *Galveston County Auxiliary* met at the home of Mrs. William T. Anderson, La Marque, on March 15 for a covered dish luncheon, and declared April 16 as Doctors' Day, when their husbands would be guests at a barbecue. Honoring their husbands on Doctors' Day, members of the *Jefferson County Auxiliary* entertained the men with a luncheon and a program, "Doctors Then and Now," in Beaumont on March 15. Early in March, the *Hunt-Rockwall-Rains-Delta Counties Auxiliary* observed a guest day with a luncheon and style show in Greenville.

*Officers of the Woman's Auxiliary to Texas Medical Association: President, Mrs. Joseph H. McCracken, Jr., Dallas; President-Elect Mrs. R. C. Bellamy, Liberty; First Vice-President, Mrs. Harold Lindley, Pecos; Second Vice-President, Mrs. William C. Barksdale, Borger; Third Vice-President, Mrs. Scott H. Martin, San Angelo; Fourth Vice-President, Mrs. L. L. D. Tuttle, Houston; Fifth Vice-Presidents, Mrs. John C. Parsons, San Antonio; Treasurer, Mrs. J. C. Terrell, Stephenville; Recording Secretary, Mrs. Franklin Campbell, Fort Worth; Corresponding Secretary, Mrs. O. M. Marchman, Jr., Dallas; Publicity Secretary, Mrs. Joe Thorne Gilbert, Austin; Parliamentarian, Mrs. O. W. Robinson, Paris.*

## AMA AUXILIARY MEETS IN JUNE

Mrs. George Turner, El Paso, will preside as President of the Woman's Auxiliary to the American Medical Associa-

tion when the group convenes June 6-10 in Atlantic City. Registration for the thirty-second annual convention will open Sunday, June 5, following a day of preconvention committee meetings. Headquarters hotel is the Haddon Hall.

The Monday program is devoted to round-table discussions on legislation, public relations, newsletters, and *Today's Health*, after which a tea will honor Mrs. Turner and Mrs. Mason G. Lawson, Little Rock, President-Elect of the National Auxiliary. A fashion show given that evening will complete the day.

The formal opening of the convention will be held Tuesday morning. During the program Mrs. Joseph H. McCracken, Jr., Dallas, President of the Woman's Auxiliary to the Texas Medical Association, will give the response to the address of welcome; Mrs. Turner, the address of the President. A luncheon and afternoon business session are scheduled. That evening the AMA convention opens, and a reception and ball in honor of the AMA President is on the agenda.

The Wednesday program will include a general meeting; a luncheon with AMA officials as guests of honor and Dr. Walter B. Martin, Norfolk, Va., President of the AMA, as guest speaker; and round-table discussions on program, civil defense, mental health, and nurse recruitment.

The annual election of officers is scheduled for the Thursday morning general meeting. Mrs. Frank N. Haggard, San Antonio, National Past President, will present the Past President's Pin to Mrs. Turner, who in turn will present the President's Pin and Gavel to the incoming President.

The annual dinner of the Woman's Auxiliary to the AMA for members, husbands, and guests will be given that evening followed by dancing.

# DEATHS

## A. Y. JENNINGS

Dr. Adolphus Young Jennings, Mabank, Texas, died in a local hospital on February 24, 1955, of a coronary occlusion.

He was the son of James V. and Cordella Jennings and was born on January 28, 1907, in Ranger. Dr. Jennings received his preliminary education at Abilene High School and Hardin-Simmons College, Abilene, and was graduated from Tulane University Medical School, New Orleans, in 1932. He interned at the Shreveport Charity Hospital. Dr.

DR. A. Y. JENNINGS

Jennings began his practice in Elysian Fields; after three years he moved to Mabank, where he had been in active practice until the time of his death.

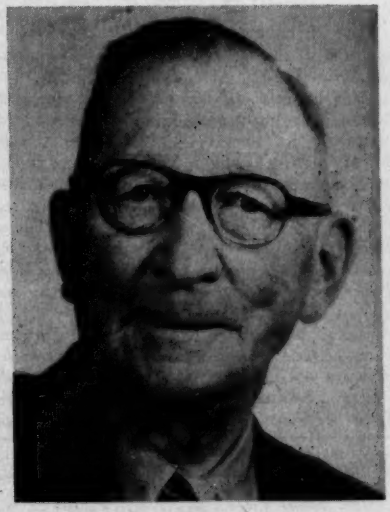
Dr. Jennings was a member of the Texas Medical Association, the American Medical Association, and the Kaufman County Medical Society of which he was a past president. He was the local surgeon for the Texas and New Orleans Railroad.

He was a Royal Arch Mason, a past patron of the Eastern Star, and a member of the Roddy Lodge. He was a member of the Baptist Church, and had served as a member of the board of trustees of the Mabank Public Schools for several years. Dr. Jennings also was an honorary citizen of Fort Worth.

Dr. Jennings and Miss Okemah Davis were married on June 25, 1933, in Shreveport, La. Mrs. Jennings died in an automobile accident December 29, 1954. Survivors include his mother, Mrs. Cordella Jennings Johns, Mabank, and two sons, James David Jennings, Mabank, and Adolphus Y. Jennings, Jr., Dallas.

## B. B. BRANDON

Dr. Ben Bernard Brandon, Edgewood, Texas, physician for forty-four years, died at his home on February 6, 1955, of arteriosclerotic cardiovascular disease.



DR. BEN B. BRANDON

Son of Ann and J. D. Brandon, he was born December 29, 1878, in Lamar County. After attending the Edgewood public school, Dr. Brandon was graduated from Baylor

*An obituary ordinarily will not be published more than four months after date of death. Cooperation in reporting deaths of physicians and in furnishing appropriate biographical material promptly is solicited.*

University and Baylor University College of Medicine. His internship was spent in Baylor Hospital, Dallas. After completing his medical education, Dr. Brandon returned in 1911 to establish his practice in Edgewood where he remained until the time of his death. He did postgraduate work in Bellevue Hospital, New York City; in the New York Polyclinic; and at Tulane University.

Dr. Brandon gained recognition for his work with malaria and had a paper on the subject published in Holland. He was a member of the Texas Medical Association, the American Medical Association, and the Van Zandt County Medical Society, which he had served as president in 1932 and as secretary from 1936 to 1955. He also was physician for the Texas and Pacific Railroad for many years and was the health officer for Van Zandt County in 1912. As local medical examiner, Dr. Brandon served during World Wars I and II and the Korean conflict.

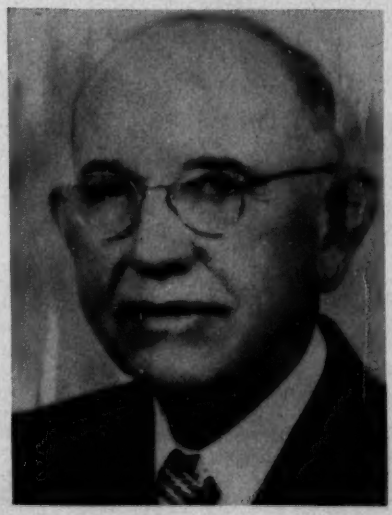
Active in civic affairs, he was mayor of Edgewood from 1934 until 1944 and served as chairman of the chamber of commerce for many years. He was a member of the Edgewood school board and donated land for the high school football field. Dr. Brandon taught the men's class in the Baptist church where he had been a member since 1888, and presented the church with a memorial organ. He also was a member of the Masonic Lodge, the Shrine, and the Lions Club.

Dr. Brandon was married October 1, 1900, to his first wife, Miss Mattie Estes, who died August 19, 1939. The former Mrs. Verlin Sherwood and Dr. Brandon were married May 29, 1941.

Survivors are Mrs. Brandon, now living in Denton; a stepdaughter, Miss Melba Sherwood, Denton, and one sister, Miss Pearl Brandon, Edgewood.

## J. A. LEGGETT

Dr. Jesse A. Leggett died at his home in Menard, Texas, on February 20, 1955, of coronary thrombosis.



DR. JESSE A. LEGGETT

Dr. Leggett was born in Little Red, Ark., November 23, 1878, to Francis Marion and Rebecca Norman Leggett. He attended preparatory school in Collin County, was graduated from the Memphis (Tenn.) Hospital Medical College in 1911, and interned at the Memphis Hospital. He practiced medicine briefly in Verona, Cash, Isom Springs in what was then Indian Territory, Rowena, and Ballinger before coming in 1915 to Menard, where he practiced until his retirement in 1949.

A member of the Texas and American Medical Associations through the Kimble-Mason-Menard-McCulloch Counties Medical Society, he also was a member of the Fourth District Medical Society. Dr. Leggett was a past president of his county society. He was county health officer for forty years. While practicing in Rowena, Dr. Leggett was appointed surgeon for the Santa Fe Railroad.

A member of the Baptist Church, he was a deacon for forty years and chairman of the board of deacons for twenty-eight years. He also had been on the local school board. Dr. Leggett had extensive ranching interests in and near Menard County.

During both World Wars, Dr. Leggett served as medical examiner for the Menard County Draft Board.

Miss Martha Sue Westbrook and Dr. Leggett were married in Greenville in 1900. She survives as do four children, Mrs. Anton Theis, Raymond F. Leggett, and Dr. L. Waldo Leggett, all of Midland, and Malcolm S. Leggett, Menard; and two sisters, Mrs. Sarah Tucker, Dallas, and Mrs. Johnnie Mack, Shreveport.

## W. W. BROOKS

Dr. William Wilkerson Brooks of Borger, Texas, pioneer physician and surgeon, died of a heart attack on March 24, 1955.

Born July 19, 1895, in Memphis, Tenn., he attended the Christian Brothers College in Memphis and was graduated from the University of Tennessee Medical School in 1920. He interned in the John Gaston Hospital, Memphis. Before coming to Texas, Dr. Brooks practiced in Henryetta and

WILLIAM W. BROOKS

Pryor, Okla. In 1927, he moved to Phillips, Texas, and operated the Phillips Hospital until June, 1949, when he moved to Borger. There, in partnership with Dr. Willard H. Smith, he opened the Brooks-Smith Clinic, in which he was active until the time of his death.

Dr. Brooks was a member of the Texas and American

Medical Associations through the Gray-Wheeler-Hansford-Hemphill-Lipscomb-Roberts-Ochiltree-Hutchinson-Carson Counties Medical Society, and was a member of the American Association of Industrial Physicians and Surgeons. He was president of his county medical society in 1932, 1938, 1947, and 1949. He also was a past president of the North Plains Hospital Staff.

He was a member of the Methodist Church, the Borger Chamber of Commerce, and the North Plains Knife and Fork Club. He had served as a director of the First National Bank, Borger, since it was opened in 1948. Dr. Brooks also was a thirty-second degree Mason and member of the Shrine. He was a veteran of World War I.

Dr. Brooks had contributed liberally to Boys Ranch, and at his death, the Dr. Brooks Memorial Fund was established with the desire that his friends donate to the fund for Boys Ranch rather than contribute flowers.

On June 15, 1926, Miss Mary Martin and Dr. Brooks were married in Henryetta, Okla.

Survivors include his wife, Mrs. Mary Brooks; one daughter, Miss Mary Ann Brooks; two sons, Bill Brooks, a student at the University of Texas, and Lt. Mike Brooks, Warren Robins, Ga.; his mother, Mrs. C. McKenna, New York; his father, W. S. Brooks, Memphis, Tenn.; and one sister, Miss Mary Helen McKenna, New York.

## R. D. GIST

Dr. Robert D. Gist, pioneer Amarillo, Texas, physician, died on March 22, 1955, in a local hospital from regional ileitis and myocardial failure.

Dr. Gist was born to Samuel Vance and Eleanor Renfro Gist on February 13, 1882, in Era, Cook County. He received his later education at the University of Texas and the University of Texas Medical Branch, from which he was

DR. ROBERT D. GIST

graduated in 1906. In that year he went to Amarillo and served as a staff member of St. Anthony's Hospital until the time of his death. He had been a member of the executive board since 1920. A memorial loan fund in Dr. Gist's honor has been established by the staff of St. Anthony's Hospital to provide financial assistance to women entering nurses training at the hospital.

He had been a member of the Potter County Medical Society almost continuously since he began his practice. He was a member of the Third District Medical Society, the Texas Medical Association, and the American Medical Association, and was a fellow of the American College of Surgeons.

Dr. Gist served in France as a captain with the Army Medical Corps during World War I. He was a member of the Presbyterian Church. He had extensive agricultural interests in and around Amarillo, and enjoyed fishing and football as hobbies.

Miss Anna Floye Strohwig and Dr. Gist were married on June 26, 1907, in Holton, Kan. Survivors are Mrs. Gist, five daughters, Mrs. W. J. Morris, Amarillo; Mrs. Charles L. Lockett, New York; Mrs. Paul R. Knupp, Amarillo; Mrs. J. W. Wheeler, Austin; and Mrs. H. C. Federer, Jr., Pampa; his stepmother, Mrs. J. M. Templeton, Pahokee, Fla.; and two brothers, R. B. Gist, Canyon, and J. V. Gist, Orlando, Fla.

## W. B. SAUNDERS

Dr. Weaver B. Saunders, Sulphur Springs, Texas, died at a local hospital on February 20, 1955, of pneumonia.

Born on October 24, 1882, in Mansfield, he was the son of Arron R. and Cynthia Ann Saunders. He received his

DR. W. B. SAUNDERS

preliminary education at Simmons College, Abilene; he then attended the University of Texas Medical Branch, Galveston, and the Memphis (Tenn.) Hospital Medical School, from which he was graduated in 1909. He had practiced in Sweetwater, Westbrook, and Brashear before locating in 1936 in Sulphur Springs. He was county health officer for two years in Sweetwater, and was local physician for two railroads.

Dr. Saunders was a member of the Hopkins-Franklin Counties Medical Society and the Texas Medical Association. He also was a member of the Christian Church. He was in military service during World War I, and served as first lieutenant.

Miss Rosa B. Conger and Dr. Saunders were married on January 10, 1912, in Galveston. Mrs. Saunders and two children, Miss Maurine Saunders, Sulphur Springs, and Maurice Saunders, San Antonio, survive.